

# **EXHIBIT 24**

UNITED STATES DISTRICT COURT  
DISTRICT OF VERMONT

Civil Action Number: 5:16-cv-0125

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JAMES D. SULLIVAN, ET AL, INDIVIDUALLY AND ON  
BEHALF OF A CLASS OF PERSONS SIMILARLY SITUATED,  
Plaintiffs,

vs

SAINT-GOBAIN PERFORMANCE PLASTICS CORPORATION,  
Defendant.

\*\*\*\*\*

DEPOSITION OF PHILIPPE GRANDJEAN, a witness  
called on behalf of the respective party, taken pursuant  
to the applicable provisions of the Federal Rules  
of Civil Procedure, before William M. Jackson,  
Professional Court Reporter and Notary Public in and for  
the Commonwealth of Massachusetts, at the Law Offices of  
Sugarman and Sugarman, 800 Boylston Street, Boston,  
Massachusetts, on October 10, 2018, commencing at 9:08  
a.m.

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## I N D E X

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PHILIPPE GRANDJEAN

DIRECT

CROSS

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(By Mr. Wolff)

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(By Mr. Whitlock)

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PROCEEDINGS

THE VIDEOGRAPHER: Good morning. We are going on the record at 9:08 a.m. on October 10, 2018. Please note microphones are sensitive and may pickup private conversations and cell phone interference. Please turn off all cellphones and place them away from the microphones. Audio and video recording will continue unless all parties agree to go off the record. This is Media Unit 1 of the Video Recorded Deposition of Philippe Grandjean in the matter of James D. Sullivan, et al versus Saint-Gobain Performance Plastics Corporation in the United States District Court, District of Vermont. 516-CV-00125.

This deposition is being held at Sugarman and Sugarman, 800 Boylston Street, Boston, Massachusetts. My name is Gail Ashton from the firm Veritext. I am the videographer. The court reporter is William Jackson from the firm Veritext. I am not authorized to administer an oath. I am not related to any party in this action nor am I financially interested in the outcome.

Now counsel and all present in the room will state their appearances and affiliations for the record.

MR. WOLFF: Bert Wolff for the Defendant.

1 MS. PASSARETTI-WU: Rachel Passaretti-Wu for  
2 the Defendant.

3 MR. WHITLOCK: James Whitlock on behalf of  
4 the Plaintiffs.

5 PHILIPPE GRANDJEAN  
6 a witness called on behalf of the respective party,  
7 having been sufficiently identified and sworn to tell  
8 the truth, was examined and testified as follows:

9 DIRECT EXAMINATION

10 BY MR. WOLFF:

11 Q. Please state your name.

12 A. My name is Philippe Grandjean.

13 Q. As a general principle, you would agree that  
14 physicians and scientific investigators should try to  
15 look at issues critically?

16 A. That sounds reasonable.

17 Q. And physicians and scientific investigators are  
18 concerned about being accurate, true?

19 MR. WHITLOCK: Objection to the form.

20 A. I can say this much as a physician and scientist.  
21 I myself is trying to be as accurate as possible.

22 Q. And as a physician and scientific investigator,  
23 do you subscribe to the principle that it is important  
24 to use accuracy and precision in your writings?

1       A. In my writings, yes. In my writings, in this  
2 opinion for example, yes.

3       Q. Do you believe that scientists should describe  
4 their methods and explain their reasoning so others can  
5 understand how the data were analyzed and how the  
6 conclusions were reached?

7               MR. WHITLOCK: Objection to the form.

8       A. This is a little too general. Because we cannot  
9 start with the principles of physics. Before we do  
10 anything. In general terms, if we are, let's say, using  
11 innovative methods, this is particularly important.

12       Q. You would agree that criticism and rigorous  
13 attempts at refutation of a hypothesis being advanced is  
14 an integral part of the scientific method; is that  
15 correct?

16               MR. WHITLOCK: Objection to the form.

17       A. That was a very long sentence. I need it again.

18       Q. You would agree criticism and vigorous attempts  
19 at refutation of the hypothesis being advanced is an  
20 integral part of the scientific method, correct?

21       A. I would say this is in accordance with Popper's  
22 Philosophy. The positivism that it is impossible to  
23 approve usually is impossible to approve a hypothesis a  
24 hundred percent. But if we fail repeatedly, on refuting

1 it, that is support for the validity of that hypothesis.  
2 That is how I would state Popper's philosophy. That is  
3 part of modern science.

4 MR. WOLFF: Mark this as Exhibit Number 1.

5 (Report was marked as Exhibit Number 1 for  
6 identification)

7 Q. Exhibit Number 1 is a copy of your report in this  
8 matter dated August 1, 2018; is that correct?

9 A. That's sort of what it looks like.

10 Q. In the first paragraph, you write that you have  
11 been asked by counsel for the plaintiffs to provide from  
12 a medical and epidemiological perspective an expert  
13 rebuttal report responding to certain opinions authored  
14 by three experts retained by Defendant Saint-Gobain and  
15 that specifically you have been asked to provide an  
16 evaluation of the human health risks associated with  
17 environmental PFOA contamination from Saint-Gobain's  
18 manufacturing and disposal operation in North Bennington  
19 and Bennington, Vermont; is that correct?

20 A. That's what it says here right on Page 1 on the  
21 top.

22 Q. On Page 3 of your report, you further state that  
23 you have also reviewed the reports of the three experts  
24 retained by Saint-Gobain to respond by Doctor Ducatman;



1 is that correct?

2 A. That's correct.

3 Q. You also state that you have benefited from  
4 access to and review of Doctor Ducatman's two expert  
5 reports as well as his expert rebuttal report; is that  
6 correct?

7 A. That's correct.

8 Q. On Page 5 of your report, you state that you  
9 concur with Doctor Ducatman's opinion concerning a  
10 medical monitoring program; is that correct?

11 A. Is that what it says here?

12 Q. In the first bullet point here.

13 A. Yes. That a medical monitoring is appropriate.  
14 Yes.

15 Q. Ultimately, on Page 71 of your report in  
16 Paragraph Number 9, you assert that Doctor Ducatman's  
17 opinions are justifiable and reasonable on the basis of  
18 current scientific and medical evidence; is that  
19 correct?

20 A. That's correct.

21 Q. What is your understanding of your role in this  
22 litigation? In other words, how do you define your role  
23 here?

24 A. This is exactly the wording that you just read

1 out loud from Page 1. The top paragraph. That, I,  
2 medical and epidemiological expert to help rebut the  
3 three expert rebuttals from the defendant.

4 Q. Is it fair to say that you have also been asked  
5 to endorse the opinions of Doctor Ducatman in this case?

6 A. Not precisely. Because that was not my task.  
7 But indirectly, I certainly have relied upon his  
8 opinion, and I think if you compare the reports, we are  
9 very much in agreement.

10 Q. Are you aware that Doctor Ducatman was deposed in  
11 this matter?

12 A. I think I heard so. I have not, I don't know  
13 about the details.

14 Q. Because I noticed that you did not list Doctor  
15 Ducatman's deposition transcript among the materials  
16 that you considered. So have you seen that before?

17 A. What's that -- no. He was deposed recently. It  
18 must have been something way back. I don't remember  
19 frankly.

20 MR. WOLFF: Mark this as Exhibit Number 2.

21 (Doctor Ducatman's Deposition Transcript was  
22 marked as Exhibit Number 2 for identification).

23 Q. Exhibit 2.

24 A. I don't remember that one.

1       A. Exhibit Number 2 is a copy of Doctor Ducatman's  
2 deposition transcript dated February 28, 2018. Would  
3 you please turn with me to Pages 37 to 39 beginning at  
4 Line 21.

5       A. Repeat that.

6       Q. 37. Line 21. I want to ask you some questions  
7 about the next two pages.

8       A. Okay.

9       Q. So you are free to sort of peruse the transcript  
10 and look along. I want to be clear that I am asking you  
11 for your own opinions. This is not a reading exercise.

12       A. Right. It is 200 something pages.

13       Q. At this point, I am only asking you about two of  
14 them.

15       A. Okay.

16       Q. Do you agree that different people drink  
17 different amounts of water on average over the course of  
18 a day?

19               MR. WHITLOCK: Objection to the form.

20       A. This agrees with EPA exposure perimeters. I  
21 would say this is true.

22       Q. Do you agree that some people will typically  
23 drink only tap water, some people will typically drink  
24 only bottled water, and some people will drink a

1 combination of both?

2 MR. WHITLOCK: Objection to the form.

3 A. I think it is difficult only to drink bottled  
4 water, because you tend to use tap water, let's say, for  
5 boiling potatoes or making gravy. It would be hard to  
6 avoid completely trying to use tap water. I don't think  
7 that is what EPA is assuming in their exposure tables.

8 Q. In terms of, if we put cooking to the side,  
9 because I will ask separate questions about cooking, do  
10 you agree in terms of drinking in terms of a glass of  
11 water, some people will typically drink only tap water,  
12 some people will drink only bottled water, and some  
13 people will drink a combination of both?

14 MR. WHITLOCK: Objection to the form.

15 A. Again, I don't think this is likely that you can  
16 completely avoid tap water. Again, I am referring to  
17 EPA's tables, and I don't think they include people  
18 drinking absolutely zero water, because you still have  
19 to make tea and coffee. It would be highly unusual for  
20 people who make tea with bottled water.

21 Q. The sources of tap water even at areas at issue  
22 in this matter can be different from house to house?

23 A. I would assume so based on -- let me hear the  
24 question again.

1 Q. The sources of tap water even in the areas at  
2 issue in this matter can be different from house to  
3 house, true?

4 A. So the sources -- are you talking about the PFOA  
5 water?

6 Q. I am talking about where the water service comes  
7 from in the home.

8 A. So whether it is community water or well water,  
9 is that what you are asking?

10 Q. Yes.

11 A. Yes. Okay. I understand that's true. That's  
12 true from the map of the community.

13 Q. Different people will use different sources of  
14 water for cooking; is that correct?

15 A. Again, what do you mean sources? Are you talking  
16 if they have pipe water in the house from outside or if  
17 they have their own well? Is that what you are  
18 referring to?

19 Q. Some people will use tap water. Some people will  
20 use water that has been filtered with activated  
21 charcoal. Some people will use bottled water. Is that  
22 correct?

23 MR. WHITLOCK: Objection to the form. Calls  
24 for speculation. You may answer.

1           A. I can't answer that. I don't know which houses  
2 have filters. So this is not part of my report. I  
3 can't guess.

4           Q. Some people will cook exclusively or primarily  
5 with tap water whereas other people will cook  
6 exclusively or primarily with bottled water; is that  
7 correct?

8                   MR. WHITLOCK: Same objection. Speculation.

9           A. It would be highly unusual to prepare food with  
10 bottled water. I have no knowledge of that existing in  
11 this community.

12          Q. One person might drink on average a glass of tap  
13 water a day whereas another person might drink on  
14 average a gallon of tap water a day; is that correct?

15          A. I don't know if one glass is a good problem  
16 anymore. Again, I think we need to refer to EDA's  
17 exposure tables and I think a gallon is a little bit on  
18 the high side unless it is in the far south. Again, a  
19 glass is very little. So these are really extremes.

20          Q. There is a continuum of the amount of water that  
21 an average person will drink over the course of a day;  
22 is that correct?

23          A. I would agree to that.

24          Q. One person might typically drink only bottled

1 water as a general rule; is that correct?

2 A. Again, I think you are really pressing the issue  
3 that I can't say that this is correct. I would say it  
4 is unlikely that a person would simply only drink  
5 bottled water. Because I refer to my comment before on  
6 tea and coffee.

7 Q. Some people will routinely use an activated  
8 carbon based water filter before using tap water where  
9 others might not, correct?

10 MR. WHITLOCK: Objection to the form.  
11 Foundation.

12 A. I could say yes, but I don't know if that applies  
13 to this community.

14 Q. Please, turn with me to Page 69 of your report.  
15 In the context of addressing medical monitoring, halfway  
16 down the page, you state that every individual has his  
17 or her own background risk of developing the diseases in  
18 question; is that correct?

19 A. That is what it says here.

20 Q. And you further state that early life exposures  
21 to PFOA will likely lead to greater risks as will  
22 exposures occurring during pregnancies or at peak ages  
23 for say cancer development?

24 A. That's correct.

1 Q. And you also state that age should be considered;  
2 is that correct?

3 A. Right. But look at it in this connection.

4 Q. Are you done with your answer?

5 A. Yes.

6 Q. I was not sure.

7 A. This is part of a whole paragraph. Yes.

8 Q. Due to obvious differences in gender and  
9 physiology, you note that monitoring blood pressure  
10 during pregnancy would not be needed for male residents  
11 screening for prostate cancer is not needed for females;  
12 is that correct?

13 A. Exactly.

14 Q. Please turn with me to Page 55 of Doctor  
15 Ducatman's deposition transcript. Beginning at the very  
16 bottom of the page at Line 25. Then carrying on to Page  
17 61. Are you there?

18 A. Among the individuals?

19 Q. Yes.

20 A. Yes.

21 Q. Let's briefly explore whether you and Doctor  
22 Ducatman are on the same page and are in agreement with  
23 one another. Again, while you are free to read along, I  
24 am asking you whether you also share these views. Okay?



1 A. Yes.

2 Q. Among the individuals residing within the areas  
3 at issue in this matter, would you expect there to be  
4 considerable individual differences as to their amount  
5 and length of exposures to PFOA?

6 A. That's true. I agree.

7 Q. Would you expect there to be individual  
8 differences as to what their blood PFOA serum levels  
9 would be?

10 A. Yes. That is reasonable. I agree.

11 Q. Would you expect there to be individual  
12 differences as to what their susceptibilities if any to  
13 PFOA might be?

14 MR. WHITLOCK: Objection to the form.  
15 Susceptibilities. Being vague and ambiguous.

16 A. If I may just refer to my own writing. Let me  
17 explain my understanding of this. That susceptibility  
18 relates to men not developing hypertension during  
19 pregnancy and issues like that I explained in my  
20 paragraph that you read before.

21 Q. As to those individual differences in exposure  
22 blood levels and susceptible if any would that be a  
23 function of a number of different variables?

24 MR. WHITLOCK: Objection to the form.

1 Vague. Ambiguous.

2 A. It is a very convoluted sentence. It is not as  
3 precise as I would explain it in science but it seems  
4 reasonable in my understanding of it.

5 Q. Let's see if we can drill down on that. Among  
6 those individual differences would be a function of  
7 among other things their ages; is that correct?

8 A. Function of duration?

9 Q. Ages.

10 A. Yes.

11 Q. Among those individual differences would be a  
12 function of among other things gender, true?

13 A. Yes.

14 Q. Those individual differences would be a function  
15 of among other things their physiology; is that correct?

16 A. What do you mean by physiology?

17 Q. Well, physiology, the makeup of their body.

18 A. I can't answer that.

19 Q. The individual differences would be a function of  
20 among other things how long they lived in the area; is  
21 that true?

22 A. Did you say differences in exposure?

23 Q. Yes. Among the individual differences in  
24 exposure, blood levels and susceptibilities would be how

1 long they lived in the area, true?

2 A. I don't like the word susceptibility, because I  
3 don't understand your --

4 Q. Let's do it this way. Among the considerable  
5 individual differences in PFOA, exposure and blood  
6 levels, those individual differences would be a function  
7 of among other things how long they lived in the area,  
8 true?

9 A. In that meaning, I understand, it is true.

10 Q. Among the individual differences and exposure and  
11 blood levels for PFOA, those individual differences  
12 would be a function of among other things their rates of  
13 daily water consumption; is that true?

14 A. I agree.

15 Q. Among the considerable individual differences in  
16 exposure to PFOA and blood levels of PFOA, the  
17 individual differences would be a function of among  
18 other things the concentrations of PFOA in the water  
19 that they drank; is that correct?

20 A. That's correct.

21 Q. And those individual differences would be a  
22 function of among other things their sources of water;  
23 is that correct?

24 MR. WHITLOCK: Objection to the form.

1           A.   What do you mean by sources?

2           Q.   In other words, where they take the water from.  
3   If they are drinking tap water, bottled water, if they  
4   are having water from activated carbon filter systems.

5           A.   Yes.   Okay.   I mean, it is so easy to understand,  
6   well, any way, my answer is yes.

7           Q.   Among the individual differences, that would also  
8   be a function of their diet and nutrition; is that  
9   correct?

10          A.   I would not say yes, because I am not sure how  
11   that would affect their uptake of PFOA or their exposure  
12   unless this is somebody eating soup every day.   So I  
13   can't answer that.

14          Q.   Among the considerable individual differences and  
15   susceptibilities if any to PFOA exposure, those  
16   individual differences would be a function among other  
17   things one's diet and nutrition; is that correct?

18                   MR. WHITLOCK:   Objection to the term  
19   considerable.

20          A.   I can't answer that.

21          Q.   The individual differences would be a function of  
22   among other things their drug and alcohol use; is that  
23   correct?

24          A.   I disagree with the way you phrase the question.

1 I can't answer it yes or no.

2 Q. How can you answer it?

3 A. If you are talking about, let's say, exposure  
4 related liver damage, I would assume that alcoholism  
5 would make a subject more vulnerable to the PFOA  
6 associated adverse affects, but I don't have any  
7 evidence to support that. So this would be a medical  
8 assumption and I am not sure this is what you are after.

9 Q. Among the individual differences would be a  
10 function of among other things body weight and body mass  
11 index; is that correct?

12 A. Again, I have the same problem in understanding  
13 it. Because body mass and obesity could be an affect of  
14 PFOA exposure. And therefore to say that obesity or  
15 body weight determines the outcome would be to say that  
16 the affect of PFOA exposure is affecting the affect of  
17 the PFOA exposure. So that sentence does not make  
18 sense.

19 Q. Please turn for me to Page 59. Line 8 of Doctor  
20 Ducatman's deposition transcript.

21 A. (Witness complies).

22 Q. Doctor Ducatman was asked this question and gave  
23 this answer under oath at his deposition. Question, and  
24 those individual differences, would be a function of

1 among other things their body weight and body mass index  
2 or BMI, correct, and his answer was one word. Yes. Do  
3 you see that?

4 A. Yes.

5 Q. Was Doctor Ducatman wrong?

6 MR. WHITLOCK: Objection to the form.

7 A. Was Doctor Ducatman what?

8 Q. Was Doctor Ducatman wrong?

9 A. At the time, he was probably correct. And at the  
10 time that he wrote his expert report that very recently  
11 I published a study that showed exactly what I just told  
12 you and Doctor Ducatman may not have been, you know,  
13 familiar with that study, and also a second study that  
14 was just published as of one or two-weeks ago.

15 Q. So when did you publish the first study that you  
16 just referenced?

17 A. I don't remember. It may have been May or June  
18 of this year.

19 Q. What was its title?

20 A. Can I -- I don't remember the title. I can tell  
21 you what it refers to. It is a project called Pounds  
22 Lost. This was a clinical controlled trial where  
23 overweight or obese individuals from Baton Rouge and  
24 Boston used different approaches to dieting. All of

1     them had calorie restriction and were therefore exposed  
2     to lose weight. And they did over a six-month period.  
3     Then they were followed for another 18 months. And  
4     during that time, I mean, they all basically used, lost  
5     the same amount of weight in the six months, but  
6     afterwards they sort of had different paths and some  
7     people essentially kept their weights low and some  
8     people increased their weight. Do you want me to go on?

9         Q. No. I believe your paper was actually published  
10     prior to Doctor Ducatman's deposition. Okay. Let's  
11     just move on.

12                 In terms of the considerable individual  
13     differences for exposure, PFOA blood levels, and  
14     susceptibilities if any to PFOA and those individual  
15     differences would be a function of among other things,  
16     one's general state of health as well as other medical  
17     conditions, correct?

18                 MR. WHITLOCK: Objection to the form.  
19     Compound question. I object to counsel's testimony as  
20     to what is considerable and what is not. You can answer  
21     if you understood the question.

22         A. Of course one's -- may I have the wording again?

23         Q. Why don't we take a look at Doctor Ducatman's  
24     deposition transcript. Page 59. Line 13. The question

1 was, and those individual differences would be a function  
2 of among other things their general state of health as  
3 well as other medical conditions; is that correct?

4 A. I would agree with Doctor Ducatman's answer,  
5 which is yes.

6 Q. Those individual differences would be a function  
7 among other things their occupational histories; is that  
8 correct?

9 A. Do you mean individual differences in exposure to  
10 PFOA?

11 Q. Yes.

12 A. There could be some occupational exposure to  
13 PFOA. I would agree to that.

14 Q. In your opinion, among these considerable  
15 individual differences and exposures, blood levels,  
16 susceptibility to PFOA, if any, is there anything else  
17 that those individual differences could be a function  
18 of?

19 MR. WHITLOCK: Objection. Vague.

20 A. Nothing that I can remember right now. I think  
21 we went through a long list already.

22 Q. Fair enough. Would you agree that one of the  
23 hallmarks of science is the requirement of valid and  
24 reliable data?



1           A. That's the way I practice research.

2           Q. In your opinion, is it important to assess all of  
3 the available data relevant to the question at hand  
4 before arriving at a conclusion?

5                       MR. WHITLOCK: Objection to the form.  
6 Vague.

7           A. When you say relevant, it has to be considered  
8 relevant from a research point of view. So in that  
9 sense, I agree with you.

10          Q. Do you agree no study can be assessed in  
11 isolation and that all evidence based literature is  
12 needed to form a valid and reliable opinion in science  
13 and medicine?

14          A. With the exception of experimental astronomy  
15 where we cannot repeat the experiment, climate change.  
16 Within the narrow sense of my work at least, I would not  
17 rely on a single study in isolation unless it is  
18 supported directly or indirectly by other evidence.

19          Q. So you would agree that all available papers  
20 should be considered in a scientific deliberation and  
21 that the selective consideration of the literature is  
22 not a scientific procedure in the area in which you  
23 worked; is that correct?

24          A. I don't like it when you say available. Because

1     you may have a different understanding of that than I do  
2     because I rely on the strength of evidence. So I am  
3     looking at the strongest evidence that is available and  
4     I would not consider the studies that don't have much to  
5     do with the conclusions that I need to consider.

6           Q. Do you agree that selective cherry picking of  
7     data is inconsistent with a valid and reliable  
8     scientific methodology?

9           A. That I would do. That's what I have seen expert  
10    witnesses do.

11          Q. I am not sure I got an answer to that. Do you  
12    agree that selective cherry picking of data is  
13    inconsistent with a valid and reliable and scientific  
14    methodology?

15          A. Right. I agree in that sense that perhaps some  
16    experts like the defense experts have their own way of  
17    selecting the evidence and they may not call it cherry  
18    picking, but I do if it is really picking the evidence  
19    in accordance with the conclusions that they apparently  
20    want.

21          Q. In your opinion, is it scientifically valid to  
22    use one hypothesis to prove another hypothesis?

23                   MR. WHITLOCK: Objection to the form.  
24    Vague. You can answer.

1           A. A hypothesis is just what it is. It is a  
2 hypothesis. So if it is still a hypothesis, you cannot  
3 rely on that when you want to draw conclusions about  
4 another.

5           Q. In scientific writings, what does the word  
6 suggests or suggestive of mean?

7           MR. WHITLOCK: Objection to the form.

8           A. That's one of the words that we try to avoid. It  
9 says that the, there is a hypothesis here, and it may be  
10 valid, but if it is valid, you would say, shows, but  
11 suggests sort of a vaguer term that doesn't indicate  
12 that the hypothesis is proven.

13          Q. Would you agree that it is important to  
14 continually re-assess one's conclusions as new  
15 information becomes available?

16          A. I would agree.

17          Q. Have you ever been wrong or mistaken about  
18 something that you believed to be true?

19          A. I was wrong when I participated in the expert  
20 panel on PFOS and PFOA on behalf of the European Food  
21 Safety Authority under the European Union because at the  
22 time, this was I believe in 2008, and I cite that report  
23 in my expert opinion, we considered that PFOS and PFOA  
24 were much less toxic than we understand they are now.

1 So I must admit I was wrong simply because we didn't  
2 have enough evidence at that time. So I had to take  
3 that back.

4 Q. When you say you had to take that back -- let me  
5 ask you a general question.

6 When you recognized that you had been wrong  
7 or mistaken about something that you had believed to be  
8 true, what do you then do in those circumstances as a  
9 general proposition?

10 A. What I do is to publish as soon as I can my new  
11 findings that cast out upon the previous conclusions,  
12 but I cannot publish a new opinion on behalf of EFSAT  
13 for example. I think they need to be redone and they  
14 are redoing it now.

15 Q. What is the ad hominem fallacy?

16 A. Ad hominem -- I think it is a legal term. All I  
17 can say is my understanding of it. It is like you  
18 blame, let's say, an expert rather than blaming the  
19 opinion expressed by that expert.

20 Q. Is it fair to say the ad hominem fallacy is a  
21 strategy whereby one attacks the character, motive or  
22 other attribute of the person making a logical statement  
23 rather than attacking the substance of the statement  
24 itself?

1 MR. WHITLOCK: Objection. Asked and  
2 answered. Objection to the form. Compound question.  
3 Confusing.

4 A. I think your explanation is just an extension of  
5 what I gave before. So I would agree.

6 Q. In your opinion, is the use of the ad  
7 hominem fallacy a proper part of scientific criticism?

8 A. Let me answer it this way, because you have to  
9 take into account that as some people may have vested  
10 interests and they, if they are being confronted with  
11 that, they may themselves call that an ad hominem attack  
12 where it really is a matter of conflicts of interest.  
13 So in that sense, I would say conflicts of interest are  
14 very important in our judgment of evidence and writings.

15 Q. What factors do you believe give rise to a  
16 conflict of interest?

17 A. I think it is preferable to refer to the  
18 definitions given like the International Group of  
19 Medical Journal Editors. Because they refer to, well, I  
20 don't know this by heart, but as a journal editor, I am  
21 familiar with these issues, and we prefer to refer to  
22 transparency so that readers can understand the  
23 situation in which the author has expressed an opinion.  
24 It may be that most readers will not see that as a

1 conflict and other readers who may understand that this  
2 was part of, let's say, a legal case that they will say,  
3 there is a conflict of interest here.

4 Q. In your opinion, does funding give rise to a  
5 conflict of interest?

6 A. It depends on the source of the funding.

7 Q. In your opinion, is funding the only thing that  
8 can give rise to a conflict of interest?

9 A. No. Again, I refer to the International  
10 Committee of Medical Journal Editors. They considered  
11 for example stock ownership and they considered the  
12 spouse's stock ownership and issues like that.

13 Q. In your opinion, can an agenda to accomplish a  
14 social or health care purpose no matter how well  
15 intentioned give rise to a conflict of interest?

16 A. I can't answer that in general. Because it  
17 depends on what you are really referring to here.

18 Q. Is it fair to say ambition to find something  
19 original is common among scientists?

20 A. I would say so.

21 Q. Would you agree that there is a certain amount of  
22 pride that comes from being the first scientific  
23 investigator to report a new finding?

24 A. I would agree.

1 Q. On Page 19 of your report, in the last paragraph,  
2 you assert that several of Doctor Mandel's PFAS related  
3 articles were published in a journal known favor to  
4 submissions from industry doctors, and on Page 21 in the  
5 carryover paragraph, you assert that some Dr. Calabrese's  
6 articles are published in journals well known to  
7 favor submissions from industry toxicologists; is that  
8 correct?

9 A. That's what I say here.

10 Q. Based simply on where Doctors Mandel and  
11 Calabrese have published their work, is it your opinion  
12 that the findings that they reported in those articles  
13 are invalid?

14 A. No. I have published in those very same journals  
15 myself.

16 Q. Does where they publish their work make their  
17 opinions in this case any less accurate or valid?

18 A. Not taken alone.

19 Q. Isn't it true that more than 80 percent of drugs  
20 discovered and developed over the past forty years have  
21 come from industry?

22 A. That I don't know.

23 Q. On Page 21 of your report, you state that  
24 according to the Web of Science, Professor Calabrese's

1 work has been cited a several thousand times over the  
2 years. However, about twenty percent of the total are a  
3 auto citation. That is, where the author cites his own  
4 work.

5 MR. WHITLOCK: Where are you on Page 21?

6 A. It is the last full paragraph on Page 21.  
7 According.

8 Q. Why if at all should the percentage of so called  
9 auto citations influence your interpretation of another  
10 expert report in this case?

11 A. It does not by itself. But it is part of the  
12 whole picture that when Professor Calabrese has achieved  
13 as many as several thousand citations, then it is  
14 unusual that twenty percent of them come from his own  
15 writings.

16 Q. How did you come up with the twenty percent  
17 figure?

18 A. If you know the website called Web of Science,  
19 what you do is to ask for the statistics of the  
20 citations and the Web of Science will provide you with a  
21 number of citations in the journals that they cover. So  
22 he may have many more quotations or citations like that  
23 and they also provide the auto citations by number. So  
24 that's very easy to do.



1 Q. Do you ever cite your prior papers in your  
2 subsequently published papers?

3 A. I do.

4 Q. Have you ever calculated or checked the Web of  
5 Science to determine what the percentage of auto  
6 citation is for your own work?

7 A. I have not checked the exact number.

8 Q. On Page 22 of your report, in the second  
9 paragraph, you cite the Ruden and Hanson paper that  
10 addresses Doctor Guzelian and related positions about  
11 evidence based causation toxicology; is that correct?

12 A. That's correct.

13 Q. Were you aware that Doctor Guzelian published the  
14 response to Rudin and Hanson?

15 A. I don't remember it by heart, but I think I saw  
16 it.

17 MR. WOLFF: Mark this as Exhibit Number 3.

18 (Clear Path Towards an Evidence Based  
19 Toxicology EBT was marked as Exhibit Number 3 for  
20 identification)

21 Q. Exhibit 3 is a paper entitled Clear Path Towards  
22 an Evidence Based Toxicology EBT. Is this the paper  
23 that you were referring to?

24 A. Yes.

1 Q. Did you read this paper, Exhibit 3?

2 A. It is a while ago. I believe I read it.

3 Q. You do not discuss or cite Doctor Guzelian's  
4 response to Rudin and Hanson anywhere in your report, do  
5 you?

6 A. I don't remember.

7 Q. It is not listed on your reference list, is it?

8 A. If you say it is not, then I am willing to  
9 believe that.

10 Q. Why don't you cite or discuss Doctor Guzelian's  
11 response in your report?

12 A. As I remember when I wrote this, there was not  
13 anything new in this response than in his original  
14 presentation of what he calls evidence based toxicology.

15 Q. Please turn to Page 36 of Doctor Ducatman's  
16 deposition transcript beginning at Line 18.

17 A. 36?

18 Q. 36. Line 18.

19 A. Okay.

20 Q. Is it fair to say all human beings are exposed to  
21 thousands of chemicals, that human beings have many  
22 infectious diseases, and that human beings are a complex  
23 product of their environment and their genetics?

24 MR. WHITLOCK: Is that a question for Doctor

1 Grandjean?

2 MR. WOLFF: Yes, that is.

3 MR. WHITLOCK: Or are you reading it and  
4 asked him if he read it correctly?

5 MR. WOLFF: It is a question for the  
6 witness.

7 A. That sounds reasonable to me.

8 Q. When considering the effects of exposure to a  
9 chemical, shouldn't one consider what is known, what is  
10 not known, and what the external risk factors are for  
11 the individual?

12 A. I would not phrase it that way. It sounds  
13 reasonable.

14 Q. Have you spoken to any of the individual  
15 plaintiffs?

16 MR. WHITLOCK: Objection.

17 A. From this case?

18 Q. From this case.

19 A. No.

20 Q. Have you examined any of the individual  
21 plaintiffs in this case?

22 MR. WHITLOCK: Objection. Outside of the  
23 scope.

24 A. No. No, I have not.

1 Q. Have you reviewed any medical records for the  
2 individual plaintiffs in this case?

3 MR. WHITLOCK: Same objection.

4 A. It was not in my -- I was not asked to do that.

5 Q. Have you reviewed any of the deposition  
6 transcripts for any of the individual plaintiffs who  
7 have been deposed in this case?

8 MR. WHITLOCK: Same objection.

9 A. I don't remember so. If it is not listed in my  
10 report, it is because I have not.

11 Q. As of the time that you issued your report, did  
12 you have any plaintiff specific information as to the  
13 average daily consumption of water by the individual  
14 plaintiffs?

15 MR. WHITLOCK: Same objection.

16 A. Not that I remember. If I didn't state that in  
17 my report, it is because I didn't have that information.

18 Q. As of the time that you issued your report, did  
19 you have any plaintiff specific information as to the  
20 percentages of tap water versus bottled water versus  
21 activated charcoal filtered water typically consumed  
22 by the individual plaintiffs?

23 A. The answer is the same.

24 Q. Is the answer no?

1           A. The answer is that I didn't have that information  
2 as long as I remember.

3                   MR. WHITLOCK: Objection to the question.  
4 Outside of the scope.

5           Q. Do you know whether the water consumption  
6 practice and patterns of the proposed class  
7 representatives are typical of the water consumption  
8 practices and patterns of absent class members?

9           A. Again, that was not in my charge and I don't  
10 remember having seen any such information.

11          Q. Were you aware that the individual named  
12 plaintiffs in this matter have submitted sworn answers  
13 to interrogatories?

14                   MR. WHITLOCK: Objection. Calls for legal  
15 conclusion. Outside of the scope.

16          A. Again, I am not aware of this.

17          Q. So you have not reviewed any of the interrogatory  
18 answers from any of the individual named plaintiffs; is  
19 that correct?

20                   MR. WHITLOCK: Objection. Asked and  
21 answered.

22          A. Because it was not in my charge. So I have not  
23 reviewed that information.

24          Q. Starting at the bottom of Page 9 of your report,

1     you refer to a study of PFOA concentrations in the blood  
2     of Bennington residents, and you note that the geometric  
3     mean was ten micrograms per liters; is that correct?

4         A.   That's correct.

5         Q.   At the end of that paragraph, you refer to the  
6     named plaintiffs and state that their blood serum values  
7     are very high and range between 24.8 micrograms per  
8     liter and 305.1 micrograms per liter?

9         A.   Yes.

10        Q.   At the low end, among the individual plaintiffs,  
11     the 24.8 micrograms per liter is two-and-a-half times  
12     greater than the ten microgram average among the  
13     Bennington residents that were tested; is that correct?

14        A.   I think that is correct, yes.

15        Q.   At the high end among the individual plaintiffs,  
16     the 305.1 micrograms per liter is thirty times greater  
17     than the ten microgram per liter average among the  
18     Bennington residents that were tested; is that correct?

19        A.   Right. But it is also in the upper five  
20     percentile. So it is within the range.

21        Q.   Are you an immunologist?

22        A.   If I'm an immunologist? No. I am not. I have  
23     not claimed to be so.

24        Q.   Are you an internist?

1 A. No.

2 Q. Are you a hepatologist?

3 A. No.

4 Q. Are you board certified in any particular field  
5 of medicine?

6 A. I decided not to go for that.

7 Q. Do you hold any special certifications in  
8 epidemiology?

9 A. There was not such a thing when I started working  
10 in this field.

11 Q. The answer is that you do not?

12 A. I don't know. I do not. I have taught people so  
13 they could get those certifications.

14 Q. Do you hold any special certifications in  
15 toxicology?

16 A. No.

17 Q. Are you licensed to practice medicine anywhere in  
18 the United States?

19 A. No.

20 Q. Have you ever been -- strike that.

21 Have you ever been licensed to practice  
22 medicine in the United States?

23 A. I have not.

24 Q. Are you currently licensed to practice medicine?

1 A. No.

2 Q. Following your graduation from medical school in  
3 January of 1974, did you ever complete a medical  
4 residency?

5 A. I got a research position right away. So I  
6 skipped it, but I did work as a physician at Mount Sinai  
7 Hospital in New York City.

8 Q. Do you regularly see patients in a clinical  
9 setting?

10 A. Only in connection with immunological studies.  
11 They are not patients. They are members of populations.

12 Q. Do you physically lay hands on those patients?

13 A. No.

14 Q. How often do you see patients in a clinical  
15 setting if at all?

16 A. That is hard to answer because I worked at Mount  
17 Sinai Hospital for two years. Most of what I did was in  
18 connection with population studies.

19 Q. When was it that you worked at Mount Sinai?

20 A. That was in 1978 and 1979.

21 Q. So a while ago?

22 A. A while ago.

23 Q. As part of your current practice, do you diagnose  
24 disease in patients?



1           A.   No.

2           Q.   As part of your current practice, do you  
3   prescribe medication or other therapies to patients?

4           A.   I do. I can. And I do. Only to my immediate  
5   family.

6           Q.   How long has it been since you regularly saw  
7   patients in a clinical setting?

8           A.   Again, I have to say I see subjects who are  
9   members of populations that we study. I do not deal  
10   with individual patients.

11          Q.   How long has it been since you regularly saw  
12   individual patients in a clinical setting?

13          A.   I would say we have to go, formally we have to go  
14   back to 1979.

15          Q.   When was the last time that you prescribed  
16   medication to or other therapies to patients who were  
17   not members of your immediate family?

18          A.   I try not to because I refer colleagues or  
19   friends to their own physician; but I do occasionally if  
20   it is a matter of urgency, I do prescribe what I believe  
21   is medically responsible. I don't remember if that is a  
22   year ago, but something in that order of magnitude.

23          Q.   How long has it been since you regularly  
24   diagnosed disease in particular patients in a clinical

1 setting?

2 A. Well, I have to go back to Mount Sinai again.

3 Q. 1978, 1979?

4 A. 1979. Yes.

5 Q. How long has it been since you regularly  
6 prescribed medications or other therapies to individual  
7 patients in a clinical setting?

8 A. I think, when you ask a question that way, I  
9 should say I did make a decision to leave clinical  
10 practice very early.

11 Q. How long has it been since you last prescribed  
12 medicines or other therapies to individual patients in a  
13 clinical setting, are we talking again the late 1970s?

14 A. Yes. Not counting my friends and family.

15 Q. How long has it been since you last used a  
16 stethoscope to listen to the hearts and lungs of an  
17 individual patient in a clinical setting?

18 A. That was a long time ago because I got some  
19 hearing problems when I was in my forties.

20 Q. So --

21 A. I would have to go back to 1979.

22 Q. So you are an epidemiologist?

23 A. I am. I am.

24 Q. Epidemiology deals with the study of diseases in

1 populations and factors associated with it, correct?

2 A. Correct.

3 Q. And clinical medicine is intended for  
4 individuals; true?

5 A. That's the way I look at it.

6 Q. Have you ever designed a population based medical  
7 monitoring program?

8 A. I have. Because I was part, I was the toxicology  
9 expert for the, I am thinking of the translation. It is  
10 like the Board of Public Health within the Minister of  
11 Health in Denmark. I was in that position for over  
12 thirty years, I believe, and helped the Board of Health  
13 on matters like this. I am not sure if we used the term  
14 medical monitoring, because it always depends on what is  
15 available already; but these were discussions that we  
16 would have in the Board of Public Health. And also  
17 before that time when I was chief of occupational  
18 medicine for the labor department, I helped decide on  
19 the new regulation on lead exposure, occupational lead  
20 exposure and that would be required of workers with such  
21 exposure in regards to medical services, blood testing  
22 that should be available to them.

23 Q. When you said that the medical monitoring depends  
24 on what is available already, what did you mean by

1       that?

2           A.   Well, the setting in Denmark is different from  
3       here, because it is a national health service.   What one  
4       would request is something that's not available already.

5           Q.   For clinical laboratory tests, their sensitivity,  
6       their specificity, positive predictive value in a  
7       population at issue for diagnosing a particular health  
8       endpoint are expressed numerically; is that correct?

9           A.   That's correct.

10          Q.   In terms of clinical laboratory tests, you do not  
11       use the terms sensitivity or specificity in the text of  
12       your report, do you?

13                   MR. WHITLOCK:   Objection.   Outside of the  
14       scope.

15          A.   I don't remember if I did.   This is not what I  
16       was asked to do.

17          Q.   In terms of clinical laboratory tests, you only  
18       use the term predictive value once in the text of your  
19       report.   Why is that?

20                   MR. WHITLOCK:   Same objection.

21          A.   It was not my primary charge.   Maybe I shouldn't  
22       have even mentioned this.   Yes.

23          Q.   As to the various health endpoints, do you know  
24       the sensitivity, specificity and positive predictive

1 value in the population at issue for each of the  
2 clinical laboratory tests that Doctor Ducatman suggests?

3 MR. WHITLOCK: Objection. Form. Compound  
4 question. Outside of the scope.

5 A. If I needed to, I could look it up. This was not  
6 what I was asked to do.

7 Q. As we sit here today, do you know the  
8 sensitivity, specificity or positive predictive value in  
9 the population for any of the clinical laboratory tests  
10 that Doctor Ducatman suggests?

11 MR. WHITLOCK: Objection to the form.  
12 Vague. Outside of the scope.

13 A. I don't know it by heart.

14 Q. Gamma-glutamyl trans peptidase, if this test is  
15 positive, what specific disease if any is this test for?

16 MR. WHITLOCK: Objection. Outside of the  
17 scope.

18 A. I'm not sure I understand that word.

19 Q. Gamma-glutamyl trans peptidase?

20 A. Gamma what? I think that is an error. I don't  
21 know. I am not sure that exists. If you mean the  
22 transfer, it is a test that is routinely used for liver  
23 function. Pepsin is a stomach enzyme. These are my  
24 abbreviations. Here it is.

1 Q. I see. On Page 72 of your report, you refer to  
2 GGT?

3 A. Yes.

4 Q. So if the GGT test is positive, what specific  
5 disease if any is that test for?

6 A. I am not prepared to answer that question because  
7 it is outside of my report; but it is usually and has  
8 been used here as an indicator of abnormal, if it is  
9 elevated, abnormal liver function.

10 Q. Do you know if it is a test of any particular  
11 liver disease?

12 A. I don't know that by heart.

13 Q. Do you know the sensitivity and specificity of  
14 the GGT test for diagnosing liver disease?

15 MR. WHITLOCK: Objection. Outside of the  
16 scope.

17 A. Again, it is not something that I know by heart.

18 Q. Have you ever looked it up?

19 A. That's a few years ago. This is part of  
20 everyone's medical education.

21 Q. So when you say a few years ago, are we talking  
22 the 1970s?

23 A. No. I checked those matters on occasion because  
24 medical knowledge is of course expanding.

1 Q. Do you know the positive predictive value of the  
2 GGT test in the Bennington population?

3 MR. WHITLOCK: Objection. Outside of the  
4 scope.

5 A. My answer is the same. I don't have any  
6 information available on that.

7 Q. Do you know whether blood testing for GGT levels  
8 is among the care that would be provided to someone that  
9 sees a doctor regularly?

10 MR. WHITLOCK: Objection. Outside of the  
11 scope. Calls for speculation.

12 A. No. I don't have that information available.

13 Q. Would you find it unusual if some of the  
14 plaintiffs in this matter have had their GGT levels  
15 routinely checked as part of receiving regular care from  
16 a physician?

17 A. I don't have any knowledge available to allow me  
18 to answer that question.

19 MR. WOLFF: Off the record for a moment.

20 THE VIDEOGRAPHER: The time is 10:10. We  
21 are off the record.

22 (Discussion off the record)

23 THE VIDEOGRAPHER: Back on the record. The  
24 time is 10:11 a.m.

1 MR. WHITLOCK: Bert, I was going to make the  
2 proffer that the plaintiffs will stipulate that we are  
3 not offering Doctor Grandjean as an expert in medical  
4 monitoring in an attempt to hopefully shortcut some of  
5 these questions that we were just going through.

6 MR. WOLFF: I appreciate that. Let's take a  
7 break.

8 THE VIDEOGRAPHER: The time is 10:12. We  
9 are going off the record.

10 (Whereupon a break was taken)

11 THE VIDEOGRAPHER: We are back on the  
12 record. The time is 10:22 a.m.

13 Q. Throughout your report, for example, on Pages 16,  
14 23, 26, and 70, you level a criticism at the defense  
15 experts for purportedly ignoring a number of prospective  
16 studies, don't you?

17 A. I believe that is what I say here.

18 Q. While your report otherwise contains many end  
19 noted citations and brackets with numbers, when you make  
20 those assertions, you do not cite, list, or end note the  
21 studies that the defense experts purportedly ignored, do  
22 you?

23 A. I thought I did. If that is something you are  
24 missing, it should be very easy to see. They don't cite



1 very many reports either of them.

2 Q. So what perspective studies do you contend the  
3 defense experts ignored?

4 MR. WHITLOCK: Objection. Vague.  
5 Ambiguous. Point to a specific place in his report if  
6 you are going to ask him questions about that.

7 Q. Page 16. You say a substantial number of  
8 perspective studies have emerged. Although this has not  
9 been acknowledged by the defense experts. What studies  
10 are those?

11 A. When I say that there is a substantial number, it  
12 is very clear, because I think that I have listed many  
13 studies in my report here, and I simply have the  
14 impression that those reports are superficial, and I  
15 don't think that I should be asked to remember those or  
16 to cite those numerous studies. It is easy to check if  
17 you want.

18 Q. Please, give me the first ten studies that you  
19 would include in that list by first named author and  
20 year of publication?

21 A. I don't think that is a reasonable request. I am  
22 sitting here with my report and without the references.

23 Q. Your references are attached to your report, are  
24 they not?

1           A. I don't know what you mean by that question.

2           Q. What I mean by the question, if we take a look at  
3 your report, which has been marked as Exhibit Number 1,  
4 Exhibit D contains your cited publications in the report  
5 ranging from Numbers 1 through 277. So among those 277  
6 citations, please, list for me up to ten that you  
7 contend are prospective studies that the defense experts  
8 ignored.

9                   MR. WHITLOCK: Objection. It implies these  
10 studies are included in Exhibit D to Doctor Grandjean's  
11 report.

12          A. I can not do that on the spot. If it is very  
13 important to you, I can do so later this month and I can  
14 report the result to counsel.

15          Q. On Page 1 of your report, you state that you are  
16 an adjunct professor at the Harvard School of Public  
17 Health?

18          A. That's correct.

19          Q. Being an adjunct professor is not a tenured track  
20 position; is it?

21          A. I was offered a tenured, full professor position  
22 at Harvard that would pay my salary and pension; but I  
23 decided not to up for that because I wanted to keep my  
24 laboratory and my cohort studies overseas.

1 Q. Being an adjunct professor is not -- strike that.

2 Being an adjunct professor is not a tenured  
3 track position, is it?

4 A. I have a ten -- not tenured track. I have a full  
5 tenured lifetime position as full professor. I decided  
6 to be adjunct at Harvard so that I would be able to work  
7 there and do my research there at my own pleasure.

8 Q. Being an adjunct professor is not a tenured track  
9 position, is it?

10 MR. WHITLOCK: Objection. Vague. Asked and  
11 answered twice.

12 A. Already said that. I chose, I was given the  
13 option, and I chose to become an adjunct professor.

14 Q. As adjunct faculty, your primary affiliation is  
15 with another institution, and in this instance, in  
16 Denmark, true?

17 A. I was named professor when I was 32 and became  
18 chairman and also have been vice dean at my medical  
19 school overseas.

20 Q. In Denmark?

21 A. In Denmark.

22 Q. And your title at Harvard is based on your title  
23 at your institution in Denmark; is that correct?

24 MR. WHITLOCK: Objection to the form.

1       A. Well, if you want the whole story. It went all  
2 the way to the chancellor's office to figure out if they  
3 could name me a full professor at Harvard because that  
4 is what they wanted. Because I preferred to maintain at  
5 least a part-time access to my own laboratory and cohort  
6 studies, Harvard said that they would have to make me an  
7 adjunct professor which I accepted.

8       Q. So you are not engaged full-time at Harvard, are  
9 you?

10      A. No.

11      Q. You do not have the same rights and privileges as  
12 the non-adjunct faculty at Harvard, do you?

13      A. I think I do.

14      Q. As an adjunct faculty member, you do not have the  
15 privilege of voting in schoolwide faculty meetings, do  
16 you?

17      A. I think I do; but I rarely can attend. It is not  
18 relevant to me.

19      Q. Do you have your own dedicated administrative  
20 staff at Harvard or do you share the administrative  
21 staff with others?

22      A. I have my own staff.

23      Q. Do you have your own dedicated office at Harvard  
24 or do you share office space with others?

1       A. This gentleman was in my office yesterday. I do  
2 have my own office.

3       Q. Do you receive a salary from Harvard?

4       A. I do.

5       Q. Let's talk a little bit about scientific  
6 methodology.

7       A. Okay.

8       Q. Can we agree the essence of science is the  
9 scientific method?

10      A. If you say so.

11      Q. Would you say so?

12      A. It depends on what you understand by method. I  
13 sort of hesitate this is always true.

14      Q. Can we agree the essence of the scientific method  
15 is hypothesis and refutation?

16      A. Hypothesis refutation?

17      Q. Yes.

18      A. I don't get that.

19      Q. The scientific method at its essence proposes a  
20 hypothesis and then attempts to refute that hypothesis?

21      A. In agreement with the -- yes.

22      Q. At the bottom of Page 22 of your report, you  
23 state that critique may be considered appropriate  
24 for highly respected experts and may appear in

1 accordance with their high methodological standards and  
2 un-remitting scepticism to the work by younger  
3 colleagues. However a narrow focus on scientific  
4 methodology is often coupled with blindness to  
5 environmental degradation and social injustices. Not  
6 surprisingly, the strategy of criticizing research  
7 methodologies has been vigorously explored by vested  
8 interests often with the purpose of manufacturing doubt.  
9 Although it may serve an educational purpose to demand an  
10 almost unrealistic quality level of research, it usually  
11 aims blocking prudent risk management while paving the  
12 way for potentially toxic chemicals to be released in  
13 the environment. Close quote.

14 Do you see that?

15 A. I see that.

16 Q. What did you mean by that statement?

17 A. I think exactly what I quoted here. If you have  
18 any questions about that statement, I am happy to answer  
19 that.

20 Q. In your view, is it inappropriate to assess  
21 whether an opinion being advanced by scientists is the  
22 product of reliable principles and methods?

23 A. I need that again.

24 Q. Sure.

1                   In your view, is it inappropriate to assess  
2 whether an opinion being advanced by a scientist is the  
3 product of reliable principles and methods?

4           A. It relies on -- it sounds reasonable.

5                   MR. WHITLOCK: Objection. The question was  
6 vague.

7           Q. Is it appropriate to assess whether an opinion  
8 being advanced by a scientist is the product of reliable  
9 principles and methods, is that appropriate or  
10 inappropriate?

11          A. Am I using the methods or is it another scientist  
12 using appropriate methods? I don't quite understand.

13          Q. Let's say a third party wants to come in and take  
14 a look at your opinions in this case. Is it appropriate  
15 for a third party to assess whether the opinions that  
16 you are advancing in this case are the product of  
17 reliable principles and methods?

18          A. Yes. That's reasonable.

19          Q. In your view, is it appropriate to assess whether  
20 one has reasonably applied principles and methods to the  
21 facts of the case?

22          A. I agree with that, too.

23          Q. Is it your position that in assessing potential  
24 environment health-related disturbances, having high

1 methodological standards or a narrow focus on  
2 scientific methodology or an almost unrealistic quality  
3 of research is inimical to prudent risk management, and  
4 doing so is tantamount for paving the way for  
5 potentially toxic chemicals to be released in the  
6 environment?

7 A. I think you are citing from what I just said. I  
8 stand by that of course.

9 Q. Is that your opinion?

10 A. That is my opinion.

11 Q. In making the assessments described in your  
12 report, did you in any way lessen your focus on  
13 scientific methodology in order to ensure a  
14 precautionary risk assessment approach?

15 MR. WHITLOCK: Objection to the form.

16 A. I did not use different principles.

17 Q. Was David Michael's article in Scientific  
18 American a plea to use less than the highest quality  
19 science possible in environmental risk management?

20 A. That would be a misunderstanding. I also -- he  
21 wrote a book about this. In addition to the Scientific  
22 American article.

23 Q. In your assessment of the defense expert reports,  
24 do you contend that any of those experts did their work



1 with the intent of producing doubt?

2 A. It is something that appears to be so. At least  
3 in my judgment.

4 Q. You are familiar with Karl Popper's scientific  
5 theory of conjecture and refutation?

6 A. Yes.

7 Q. Do you disagree that refutation or theory  
8 falsification should be used sparingly in matters of  
9 environmental health?

10 A. What I agree is that we should use, you know, the  
11 strongest possible scientific methods to the extent  
12 possible.

13 Q. So you say to the extent possible. What does  
14 that mean?

15 A. It means, for example, that we can't achieve the  
16 highest level of scientific proof by dispensing PFOA to  
17 experimental subjects. That is unethical and illegal.

18 Q. On Page 14 of your report in the first full  
19 paragraph, you state that observational studies will  
20 rarely if ever provide a one-hundred percent proof. Do  
21 you see that?

22 A. I see that, yes.

23 Q. Is there such a concept as 75 percent proof or  
24 fifty percent proof?

1 A. No.

2 Q. As used in the concept of your report, what is  
3 your definition of proof?

4 A. We don't use that term regularly but what we say  
5 is that the evidence is convincing or not convincing.

6 Q. How do you know when something has been proven?

7 A. As I said before, it is not a term that we would  
8 regularly use because you can always raise doubt which  
9 means that the perfect proof does not exist.

10 Q. Let's turn for a moment to words that you  
11 regularly use. The word association is not a substitute  
12 for the word causation; is it?

13 A. That's correct.

14 Q. In fact, Doctor, wouldn't you agree that  
15 distinguishing between concepts of causation and  
16 association is an important area of scientific  
17 discussion?

18 A. Yes. I would agree so in general terms.

19 Q. Wouldn't you agree that a number of different  
20 types of relationships may exist between an exposure and  
21 an outcome and one type may be a spurious association  
22 resulting from chance bias or confounding?

23 A. It is possible.

24 Q. Would you agree that three general categories of

1 phenomena can result in an association found in a study  
2 to be erroneous, chance, bias and confounding?

3 A. It is possible.

4 Q. Does a statistically significant finding in a  
5 reliable epidemiological study that looks at the disease  
6 risk as a function of exposure necessarily mean that  
7 there is a causal relationship?

8 A. That is correct and I assessed that in my report.

9 Q. When you say it is correct, are you saying that a  
10 statistically significant association means that there  
11 is a causal relationship?

12 A. No. It is an issue that one has to consider  
13 whether it exists. It is not a necessary condition.

14 Q. Would you agree that if bias or confounding  
15 affects a study, it can invalidate an association that  
16 the study found even if it was a statistically  
17 significant association?

18 A. That is always possible.

19 Q. Can confounders be both known and unknown?

20 A. That is correct.

21 Q. There can be known and unknown sources of bias in  
22 an epidemiological study; true?

23 A. I believe so.

24 Q. When you read the report of an epidemiological

1 study, what factors do you consider in order to  
2 determine whether the results are supportive of a cause  
3 and effect relationship between the exposure and  
4 disease?

5 A. I think you mentioned at least most of the  
6 factors that I would look at.

7 Q. You would agree that the statistical significance  
8 test is a test of the data and not of the hypothesis  
9 being advanced; is that correct?

10 A. That's correct.

11 Q. In a study with a P value of 0.05, if the 95  
12 percent confidence interval includes one, then the data  
13 are not statistically significant for that finding, are  
14 they?

15 A. That is a correct interpretation.

16 Q. Epidemiological studies never establish causation  
17 in a particular person, do they?

18 A. In a single person, no.

19 Q. What is a cross sectional study?

20 A. A cross sectional study is one where all the  
21 relevant perimeters are obtained at the same time.

22 Q. So cross sectional studies examine both the  
23 exposure of interest and individuals with or without the  
24 disease of interest at a single point in time; is that

1 correct?

2 A. That's correct.

3 Q. While cross sectional studies can determine the  
4 prevalence of disease, they do not determine the  
5 incidence or risk of disease; true?

6 A. I would not say so in general, because it depends  
7 how cross sectional it is if you are just looking at  
8 exactly the status today; but, maybe, you know, a time  
9 varying of information that you are obtaining cross  
10 sectionally. So I can't say that this is always true.

11 Q. What are the limitations of cross sectional  
12 studies?

13 A. What are verifications?

14 Q. No. What are the limitations of cross section  
15 studies?

16 A. I think you mentioned the time factor. That is  
17 certainly important because in a study like this, you  
18 would like the exposure to occur before the outcome in  
19 order to support, possibly support hypothesis of  
20 exposure being hazardous.

21 Q. In cross sectional studies, because both exposure  
22 and disease are determined at the same point in time,  
23 such studies do not demonstrate that exposure precedes  
24 the disease, do they?

1       A. Again, I hesitate to say yes, because if we talk  
2 about PFOA, we are here and if you mean the exposure in  
3 terms of the concentration in the water in the  
4 household, I would say the concentration of the water,  
5 let's say, it has changed over time. For example,  
6 because a filter has been installed. Then it would be  
7 wrong to associate the concentration of PFOA in the  
8 water exactly today with the health status of that  
9 household. On the other hand, if you talk about blood  
10 PFOA concentrations, then they are a result of  
11 accumulations over time and it is not a typical cross  
12 sectional study for that reason. Because the blood  
13 concentration reflects what has happened over several  
14 years in the past. Therefore, one should not just  
15 ignore that information because it has to do with  
16 accumulation and then perhaps it may be diseases where  
17 the actual status today is also something that has been  
18 established over time. So it is not a typical example.

19       Q. Merely because a person lives in an area for,  
20 let's say, one year, that has PFOA in the water system  
21 does not tell you how much PFOA is in their blood serum,  
22 does it?

23       A. It tells me that the concentration will be  
24 elevated. It does not tell me the exact number if that

1 is what you are after. It does not tell me the exact  
2 number.

3 Q. And the extent to which the concentration might  
4 be elevated would be a function of the amount of PFOA in  
5 the water system; is that correct?

6 A. Right. There is no doubt that it will be  
7 elevated.

8 Q. It is also a function of how much water an  
9 individual drinks a day, for example, if an average  
10 person, if a person on average drinks one glass a day  
11 and another person drinks five glasses a day, their  
12 blood serum levels are likely to be different; true?

13 A. Yes, but they will all be elevated. This is also  
14 what the state found.

15 Q. So, for example, in this case, among the  
16 individual named plaintiffs, we have PFOA blood serum  
17 values ranging from roughly 28 micrograms per liter all  
18 the way up to 305 micrograms per liter; is that correct?

19 A. That's what we went through before.

20 Q. The geometric mean in the Bennington area for  
21 the people tested was 10.1 micrograms per liter?

22 A. Give me that number again.

23 Q. 10.1.

24 A. I think so, yes.

1 Q. So there is a wide range of blood serum levels?

2 A. Yes.

3 Q. Because cross sectional studies measure exposures  
4 and health conditions simultaneously, isn't it fair to  
5 say in cross sectional studies, the temporal  
6 relationship between an exposure and a disease cannot be  
7 inferred?

8 A. I need to clarify this because, as I said before,  
9 if the exposures are a matter of the consumed  
10 concentration of PFOA, it does go years back and  
11 therefore you cannot just conclude that it is a typical,  
12 cross sectional study where everything is measured at  
13 the same time and therefore it is not as strong. In  
14 this case with PFOA, it is actually stronger than a  
15 typical cross sectional study.

16 Q. Why is that?

17 A. Well, because I explained before that the  
18 concentration of PFOA that you have in your serum today,  
19 that is a result of your exposure in recent years.  
20 Therefore, it is not just something that you get right  
21 today. This is your build up in your body and therefore  
22 a longer term than just today.

23 Q. Is it fair to say that cross sectional studies  
24 are rarely useful in identifying toxic agents?



1           A. I think that is an overestimation for the reasons  
2 I just told you. In fact, many cross sectional studies  
3 actually underestimate the degree of a hazard simply  
4 because the association is more or less dwarfed by  
5 noise. Statistical noise and variations. Therefore,  
6 they are not as strong as prospective studies of course.

7           Q. What is risk?

8           A. Risk is the likelihood of an outcome, a  
9 probability if you want.

10          Q. What's the difference between relative risk and  
11 absolute risk?

12          A. Well, the relative in comparison to some  
13 reference population say where the absolute risk is the  
14 actual risk that the population of interest has.

15          Q. What is absolute risk?

16          A. Well, how should I explain this to you. It is a  
17 likelihood of this unwanted outcome.

18          Q. When considering risk, are dose and the hazard  
19 important factors?

20                 MR. WHITLOCK: Objection to the form.  
21 Vague. You can answer.

22          A. Insofar as we are relating that risk to a  
23 predictor.

24          Q. What do you mean by a predictor?

1       A. A predictor would be a hazard. A suspected  
2 hazard.

3       Q. Biological plausibility, when we say something is  
4 biologically plausible, that simply means it makes  
5 biological sense; isn't that true?

6       A. That is a nice way of putting it.

7       Q. You would agree biologic plausibility is not  
8 synonymous with causation, wouldn't you?

9       A. It is something that you take into regard when  
10 you judge whether or not there is causation.

11       Q. Biological plausibility itself is not sufficient  
12 to establish causation; true?

13       A. One would never do that. It is an irrelevant  
14 question. I am sorry.

15       Q. Does the dose response relationship stand for the  
16 proposition that the greater the dose, the greater the  
17 likelihood for the effect?

18       A. That's reasonable, yes.

19       Q. Is it fair to say that it is not only important  
20 to know that a substance is capable qualitatively of  
21 causing something, but that it is also important to know  
22 the quantitative eligibility of the suspected agent?

23       A. It is if possible.

24       Q. And isn't there a classic saying that everything

1 is toxic and it is just a matter of dose?

2 A. Yes. That's what Paracelsus said three hundred  
3 years ago. That you will see among my references that I  
4 have more recently of course adjusted that statement  
5 because it is kind of a blessing.

6 Q. In sufficient doses, table salt can be quite  
7 toxic; is that true?

8 A. If you want to call it toxicity, you can claim  
9 that you died from eating an enormous amount of salt. I  
10 would not consider that toxicity as such.

11 Q. Oxygen can be toxic?

12 A. If you want.

13 Q. What is a threshold?

14 A. Well, a threshold is the issue that an exposure  
15 in this case below a certain level does not represent a  
16 hazard.

17 Q. You would agree that cigarette smoking can cause  
18 lung cancer, correct?

19 A. I would agree.

20 Q. You would not consider smoking one cigarette in a  
21 lifetime to pose a significant risk of lung cancer,  
22 would you?

23 A. Not one cigarette alone. No.

24 Q. If someone did develop lung cancer after smoking

1 one cigarette, would you conclude that there was a  
2 significant probability that it was caused by the one  
3 cigarette?

4 A. No, I would not.

5 Q. Do we agree that a temporal relationship does not  
6 establish causation, in other words, just because a  
7 condition follows an exposure, it does not necessarily  
8 mean that the exposure caused the condition, does it?

9 A. No. It is like, these issues you could say they  
10 are nonreciprocal in that it is important to know that  
11 is the case, that the exposure comes before the outcome,  
12 but the fact that it does not prove anything.

13 Q. When one considers the effects to exposure to a  
14 chemical, isn't it important to distinguish between that  
15 effects have been reported and those which are merely  
16 feasible or theoretically possible?

17 MR. WHITLOCK: Objection to the form.

18 A. I don't understand feasible. What do you mean?

19 Q. Okay. Let me modify the question.

20 When one considers the effects of exposure  
21 to a chemical, isn't it important to distinguish between  
22 effects that have been reported and those which are  
23 merely theoretically possible?

24 A. That makes sense.

1 Q. Let's talk a little bit about animal data. Isn't  
2 it fair to say that extrapolating data from animal  
3 models to humans can be fraught with difficulties?

4 A. Yes. I would agree.

5 Q. Isn't it fair to say that attempts to extrapolate  
6 data from animals to humans must be done with caution?

7 A. That's correct.

8 Q. Isn't it fair to say that among other things  
9 there can be interspecies differences in metabolic  
10 rates, anatomy, cellular biochemistry and the absorption  
11 distribution metabolism and the elimination of  
12 chemicals?

13 A. They can be, yes.?

14 MR. WHITLOCK: Objection to the form.

15 Q. Wouldn't you agree the results of an animal study  
16 will be influenced in part by which species of animals  
17 are used?

18 A. That's correct.

19 Q. Wouldn't you agree that even among experimental  
20 animal models, different species can exhibit different  
21 sensitivities or susceptibilities or reactions to  
22 various substances?

23 A. I agree.

24 Q. Isn't it fair to say that even different strains

1 of the same species sometimes experience different  
2 reactions to the same substance?

3 A. I would agree.

4 Q. Let's talk a little bit about the ATSDR which you  
5 referenced a bit in your report.

6 A. Yes.

7 Q. A what is the ATSDR?

8 A. The Agency for Toxic Substance and Disease  
9 Registry.

10 Q. The ATSDR is a part of the Federal Centers for  
11 Disease Control and Prevention; is that correct?

12 A. That's correct. And I am going there tomorrow.

13 Q. Nice. Where is that?

14 A. In Georgia. It is in Atlanta.

15 MR. WOLFF: Mark this as Exhibit Number 4.

16 (Copy of the August 2015 ATSDR was marked as  
17 Exhibit Number 4 for identification)

18 Q. Exhibit 4 is a copy of the August 2015 ATSDR, CDC  
19 tox guide for per perfluoroalkyls. Have you seen this  
20 document before?

21 A. Yes.

22 Q. Please turn with me to the second page. In the  
23 right-hand column in the second bullet point, are you  
24 there?

1           A.    Okay.

2           Q.    The CDC states that the primary effects observed  
3           in animals include liver toxicity, developmental  
4           toxicity, and immune toxicity. There are profound  
5           differences in the toxicokinetics and mode of action of  
6           perfluoroalkyls between humans and experimental animals.  
7           Many of the observed affects in animals result from the  
8           ability of PFOA and PFOS to activate perisome  
9           proliferator activated receptor alpha. PPAR. Alpha.  
10          Humans are much less responsive to PPAR alpha than  
11          rodents and thus may not be as susceptible to these  
12          types of effects. Do you see that language?

13          A.    I see that.

14          Q.    Do you agree with that statement?

15          A.    No.

16          Q.    Why not?

17          A.    Because this is a statement that was prepared in  
18          2015, and that was from a draft toxicology profile from  
19          ATSDR and they updated that in 2018.

20          Q.    Is there an updated tox guide for perfluoroalkyls  
21          from ATSDR CDC?

22          A.    I don't remember.

23          Q.    When you say they updated that in 2018, what did  
24          they update?

1 A. They updated the tox profile.

2 Q. Is what you are referring to a several hundred  
3 page document?

4 A. That's correct.

5 Q. Let's talk a little bit about regulatory standard  
6 setting. Okay?

7 A. Okay.

8 Q. In the regulatory context where public safety and  
9 health policies are at issue, is an assumption of  
10 causation often made as opposed to a clinical setting  
11 where the criteria for causation are more stringent?

12 MR. WHITLOCK: Objection to the form.

13 A. I don't like the wording of that question because  
14 in a clinical setting, you do not need to have foolproof  
15 of a causation before you do this appendectomy. So you  
16 need to clarify what you mean.

17 Q. Are there different standards for evaluating  
18 causal hypotheses when one deals with matters of  
19 regulatory public health policy as opposed to hypothesis  
20 testing in experimental science?

21 MR. WHITLOCK: Objection to the form.

22 A. I am not sure I can answer that question.

23 Q. Is it a basic tenet of regulatory public health  
24 policy to in effect err on the side of caution?



1           A. I would say so.

2           Q. Do you agree that the government may regulate or  
3 indeed prohibit the use of a chemical despite the low  
4 probability of a causal relationship?

5                   MR. WHITLOCK: Objection to the form.  
6 Vague. Ambiguous.

7           A. I would say you use the word may. So I have to  
8 say it is possible, but I am not sure I ever saw that  
9 happening.

10          Q. The manner in which one errs on the side of  
11 caution in establishing regulatory public health policy  
12 is not something that one has to do in a basic science  
13 environment, is it?

14          A. When you say so. I would think that is not what  
15 you want to do because no science is perhaps a major  
16 input that the regulatory agencies would want.

17          Q. Is there any reason to err on the side of caution  
18 in a toxic tort suit where regulatory public health  
19 policy is not at issue but rather than scientific  
20 reliability?

21          A. I can't answer that question. It sounds to me  
22 like it is a legal question.

23          Q. What is the precautionary principle?

24          A. There are various definitions of precautionary

1 principle. One of them is from the United Nations.  
2 Another one is from a European commission. There is one  
3 from the European Environment Agency, but I can  
4 paraphrase if you want.

5 Q. I know you have written on this subject a bit.

6 A. I have.

7 Q. How do you use the word precautionary principle,  
8 what's your preferred definition of the term?

9 A. Right. I have to clarify. I don't use it  
10 myself. I can discuss what it may mean in regard to  
11 interpreting science. I can enter that discussion.

12 Q. Okay. Please, have at it.

13 A. So the precautionary principle essentially says  
14 that in the face of uncertainty, one should not allow a  
15 highly suspected hazard to continue if there are  
16 substantial risks associated with that.

17 Q. The precautionary principle can be summed up with  
18 the axiom that it is better to be safe than to be sorry;  
19 is that correct?

20 A. I think that is a very popular term. I would not  
21 express it that way myself. It relates to what you just  
22 said.

23 Q. Though you might not express it that way  
24 yourself, that's a fair summary; is that correct?

1 A. No.

2 Q. Why not?

3 A. Because it is too far fetching to say better safe  
4 than sorry and it is too loose. I don't know what you  
5 mean. What do you mean by saying safe, what do you mean  
6 by sorry?

7 Q. Does the precautionary principle inform the  
8 premise for regulatory public health policy?

9 A. I don't think so.

10 Q. The precautionary principle does not apply to  
11 clinical medicine, does it?

12 A. The way that the European Environment Agency has  
13 fine tuned the definition, it is not in regard to  
14 preventive medicine, I mean to clinical medicine, but I  
15 would say that when a physician judges that child may  
16 have appendicitis and we better operate on that, you  
17 could say that is an application of the precautionary  
18 principle.

19 Q. Was your opinion in this case informed by the  
20 precautionary principle?

21 A. No.

22 Q. On Page 63 of your report, you note that the  
23 untested chemical assumption is a common concern in risk  
24 assessments; is that correct?

1 A. That's correct.

2 Q. What is the untested chemical assumption?

3 A. The wording comes from the National Research  
4 Council in this country. The wording means that it is  
5 not logical and responsible to assume that an untested  
6 hazard has absolutely no affects. Meaning there is no  
7 risk associated with it.

8 Q. Is your criticism of the untested chemical  
9 assumption rooted in the precautionary principle?

10 A. I think those are two different matters.

11 Q. The EPA's health advisory for PFOA in drinking  
12 water is 70 parts per trillion or PPT?

13 A. Yes.

14 Q. The Vermont Department of Health has set its  
15 drinking water advisory limit for PFOA at twenty PPT; is  
16 that correct?

17 A. That used to be correct, because now that limit  
18 has changed to be the sum of PFOA and other major P  
19 facets.

20 Q. In your opinion, these advisory levels are too  
21 high; is that correct?

22 A. That's correct.

23 Q. You estimate that the exposure limits should be  
24 less than one part per trillion, is that correct?

1 A. Yes.

2 Q. You arrived at this proposed exposure limit based  
3 on a reported association between PFOA exposure and  
4 antibody concentrations?

5 A. That is the evidence that we put forward.

6 Q. At the top of Page 6 in your report, you wrote  
7 PFOA concentrations in drinking water are known to  
8 correlate with the serum concentrations of long term  
9 residents in Ohio and West Virginia at an approximate  
10 ratio of one to one hundred. Do you see that?

11 A. I don't see it, but I think this is correctly  
12 quoted.

13 Q. Using that ratio then, one part per trillion of  
14 PFOA in drinking water would be equivalent to  
15 approximately one hundred parts per trillion of PFOA in  
16 the blood serum of an individual consuming that water;  
17 is that correct?

18 MR. WHITLOCK: Objection to the form.

19 A. I am not sure I follow your calculations. If you  
20 talk about the ratio we just talked about, then I would  
21 agree with you on average.

22 Q. Just in terms of sort of leveling the various  
23 units of measure, one hundred parts per trillion is  
24 equivalent to 0.1 micrograms per liter; is that correct?

1           A. I think so.

2           Q. Is it your opinion that medical monitoring would  
3 be reasonably necessary for individuals with PFOA  
4 exposure greater than 0.1 micrograms per liter?

5                   MR. WHITLOCK: Objection. Outside of the  
6 scope.

7           A. This is not what I stated. I stated it would be  
8 appropriate for those with an elevated exposure to PFOA.  
9 So this is not in accordance with what I wrote in my  
10 report.

11          Q. Elevated in comparison to what?

12          A. First of all, in comparison with that subject's  
13 background. I think that is what this report is about.  
14 Because they have been exposed to contaminated drinking  
15 water. I am saying, it is the elevation that follows  
16 from that exposure that then leads to the recommendation  
17 of medical monitoring.

18          Q. So over the years, NHANES has reported the  
19 geometric means of PFOA in blood serum in Americans that  
20 have been tested, and so I think that in your report at  
21 Page 10, you make note that the current background  
22 levels of PFOA in blood serum that the geometric mean is  
23 2.1 micrograms per liter based on the overall US  
24 population 2011 to 2012?

1 A. That's correct. I see that.

2 Q. So is it your opinion then that an elevated blood  
3 serum level for PFOA is something greater than 2.1  
4 micrograms per liter?

5 A. No. That is not what I say in my report. I  
6 refer to the exposure. This is a population that is  
7 exposed to PFOA from Saint-Gobain in their drinking  
8 water and that leads to an elevated exposure.

9 Q. Elevated as compared to what?

10 A. To compare to not being exposed to that drinking  
11 water. That is drinking water without PFOA  
12 contamination in it.

13 Q. So would you consider that if a person's blood  
14 serum levels had a contribution of 0.1 micrograms per  
15 liter from PFOA coming from the tap water, that that  
16 would be an elevation as you used the term?

17 A. I don't quite get the numbers. What we talk  
18 about here is a substantial contamination of the  
19 drinking water, and where people that live there, and  
20 residents, not just people visiting, but residents have  
21 been exposed to this, that leads to an elevation.

22 Q. What would you be consider to be quantitatively a  
23 substantial elevation of PFOA blood serum levels against  
24 what it would have been had there not been those

1 exposures?

2 A. Right. I mean, you can read my paper from 2013  
3 where we calculate that the safe level should actually  
4 be about or below 1 PPT. And I stand by that. So  
5 meaning that an elevation, I mean, we talked about a  
6 manmade compound that does not serve any purpose in  
7 drinking water. That these people unwittingly have been  
8 drinking for years. I would say that results in an  
9 elevation because that substance is not meant to be  
10 there.

11 Q. I am asking what quantitatively is the elevation  
12 in blood serum that you would consider to be a  
13 substantial elevation as you used the term?

14 A. I understand. I have not calculated what that  
15 might mean, but I refer to the drinking water  
16 concentrations, and I have seen the map of all those  
17 wells in the community water and it is very clear that  
18 they are substantial elevations and exposure.

19 Q. Well, since the background blood levels of PFOA  
20 in the United States exceeds 0.1 micrograms per liter,  
21 is it your opinion that every American should be  
22 eligible to receive medical monitoring for that  
23 exposure?

24 A. Okay. That's a misunderstanding.



1 Q. I am asking a question.

2 A. No. That is not my opinion.

3 Q. Okay. That was my question. Is that your  
4 opinion?

5 A. Right. My opinion is that is not the case. This  
6 is a very specific case with contaminated drinking  
7 water. I know the 2.1 is part of the classification,  
8 what do you call it, the class definition. That's part,  
9 I understand that.

10 MR. WOLFF: Off the record for a second.

11 THE VIDEOGRAPHER: The time is 11:12. Off  
12 the record.

13 (Discussion off the record)

14 THE VIDEOGRAPHER: We are back on the  
15 record. The time is 11:13.

16 Q. In this case, are there substantial elevations of  
17 PFOA in the community water supply or is that limited to  
18 just private wells?

19 A. As I recall the numbers, it is in particular in  
20 certain private wells.

21 Q. While everything is toxic at some dose, the  
22 exposure levels calculated in a regulatory risk  
23 assessment do not tell you what that dose is, do they?

24 A. Let me try to rephrase the way I understand it.

1 That the regulatory guidelines or whatever they are  
2 refer to particular water concentrations and they cannot  
3 exactly be translated into serum levels or serum  
4 concentrations or individual disease risks.

5 Q. Do you consider yourself an expert on the  
6 regulatory and administrative procedures for setting an  
7 exposure limit?

8 A. I would leave that to others to decide. I have  
9 certainly advised regulatory agencies on which decisions  
10 to reach.

11 Q. In the context of regulatory risk assessments and  
12 the setting of regulatory exposure levels, what are  
13 uncertainty factors?

14 A. Well, uncertainty factors they are applied when  
15 translating dose levels that have been applied in animal  
16 studies to levels that may be considered virtually safe  
17 in humans.

18 Q. Does the EPA apply uncertainty factors to account  
19 for the extrapolation of results in experimental animals  
20 to humans, inter individual variability including  
21 sensitive subgroups, extrapolation from a lowest  
22 observed adverse effect level to a no observed adverse  
23 affect level, extrapolation of results from subchronic  
24 exposure to chronic exposures and database inadequacies?

1 A. That is my understand.

2 Q. A risk assessment may incorporate multiple  
3 uncertainty factors to calculate a regulatory exposure  
4 level; is that correct?

5 A. That's my understanding.

6 Q. In the past, uncertainty factors have been  
7 described as safety factors; is that correct?

8 A. That's correct.

9 Q. Given the methods, assumptions, and uncertainty  
10 factors that are used in regulatory risk assessments,  
11 the permissible exposure levels that are calculated are  
12 intended to be protective not predictive?

13 A. No. They are intend to be virtually protective  
14 for the exposed population.

15 Q. Virtually protective?

16 A. I think we use the word virtually because you can  
17 never completely exclude, that some adverse affect may  
18 happen.

19 Q. Given the methodology, assumptions and safety  
20 factors that characterize regulatory risk assessments,  
21 the permissible exposure levels do not provide  
22 predictive information about actual clinical risks for  
23 exposures that exceed such levels, do they?

24 A. No.

1 Q. And given the methodology assumptions and safety  
2 factors that characterize regulatory risk assessments,  
3 the permissible exposure levels do not provide  
4 predictive information about medical causation for  
5 exposures, that exceed such levels, do they?

6 A. I don't fully agree with you because medical  
7 causation is probably part of the risk assessment made  
8 by that. I mean depending on the circumstances of the  
9 agency. So one is of course trying to define a  
10 protective limit or guideline so that medical causation  
11 would not be a problem.

12 Q. On Page 4 and elsewhere throughout your report,  
13 you note that your opinion was formed by using a weight  
14 of the evidence approach; is that correct?

15 A. Weight of the evidence, correct.

16 Q. What is a weight of the evidence approach?

17 A. This is the approach that is commonly used by  
18 regulatory agencies in regard to weighing, using that  
19 term, the evidence in regard to whether it is reliable  
20 and solid and can be applied to in the risk assessment  
21 process.

22 Q. How if at all does a weight of the evidence  
23 approach differ from Global Introspection?

24 A. From Global?

1 Q. Introspection?

2 A. Introspection?

3 Q. Yes,

4 A. I don't understand the term in this regard.

5 Q. At the bottom of Page 4 of your report, you  
6 criticize the defense experts for using causality  
7 criteria that are not in accordance with weight of the  
8 evidence procedures, applied by regulatory agencies and  
9 international organizations; is that correct?

10 A. Correct. That's what I said.

11 Q. On Page 19 of your report, in the second  
12 paragraph, you again criticize the defense expert  
13 reports and write, this report's, referring to your  
14 report, this report's previous sections described proper  
15 evaluation of toxicity risks as used by regulatory  
16 agencies and international organizations like the World  
17 Health Organization; is that correct?

18 A. That's correct.

19 Q. And in sum, on Page 70 of your report in the  
20 second numbered paragraph, you state that the defense  
21 experts rely on strict criteria for causality rather  
22 than an assessment of the weight of the evidence as  
23 applied by agencies such as ATSDR, EPA, NTP, EFSA and  
24 IARC, is that correct?

1 A. That's correct.

2 Q. Does the weight of the evidence assessment you  
3 performed in this litigation conform to the approaches  
4 of the ATSDR, EPA, NTP, EFSA, and IARC in conducting  
5 risk assessments?

6 A. I believe so.

7 Q. Is it fair to say that regulatory and advisory  
8 bodies, such as IARC, OSHA, and EPA, utilize a weight of  
9 the evidence method to assess the carcinogenicity of  
10 various substances in human beings and suggest or make  
11 prophylactic rules governing human exposure?

12 A. I believe so.

13 Q. Is it also fair to say that regulatory and  
14 advisory bodies such as ATSDR, EPA, NTP, and EFSA  
15 utilize a weight of the evidence method to assess  
16 noncarcinogenic endpoints of various substance in human  
17 being and suggests or make prophylactic rules governing  
18 human exposure?

19 A. I believe so.

20 Q. Is it fair to say that this methodology results  
21 from the preventive perspective the agencies adopt in  
22 order to reduce public exposure to harmful substance?

23 A. That I'm not sure I can answer.

24 Q. Why is that?

1 A. I am a scientist. Is not a regulator.

2 Q. What is confirmation bias?

3 A. Confirmation bias?

4 Q. Yes.

5 A. Well, I mean, I am not sure I can define it here.

6 Q. Let me ask you this. Confirmation bias can arise  
7 when individuals tend to interpret, focus on, and  
8 remember information in a way that conforms to their  
9 preconceived notions; is that correct?

10 MR. WHITLOCK: Objection to the form.

11 A. I don't think that is a reasonable definition.

12 Q. Confirmation bias can affect scientific  
13 decisionmaking, true?

14 A. It is a theoretical possibility.

15 Q. Conclusion driven science is not really science  
16 at all, is it?

17 A. I disagree with that.

18 Q. Why?

19 A. Because decisionmaking in science, as I see it,  
20 it's a product, it follows from an original examination  
21 of the question, and, therefore, it's an integral part  
22 of science as you try to draw conclusions.

23 Q. Conducting science to conform with one's  
24 preconceived notions is not really science at all, is

1 it?

2 A. If you can call it science. But I would say it's  
3 biased if it happens the way you describe it.

4 Q. Where if at all in your report do you explain how  
5 a weight of the evidence approach is implemented?

6 A. I believe that I have done that in a whole  
7 section of my report.

8 Q. Can you please point me to that?

9 A. Okay. Methodological aspects, and risk evaluation.  
10 It starts on Page 11. You have to take the next  
11 section, Section 4, knowledge, you have to consider that  
12 as well.

13 Q. Maybe you can point us to the pages where the  
14 discussion appears in your report.

15 A. Right. 11 and onwards to 15 and perhaps even up  
16 to 19.

17 Q. What objective standards, if any, did you use to  
18 assign differing weights to different data sets?

19 A. Exactly the standards that I have described in my  
20 report in that I consider the power, the statistical  
21 power of the study, magnitude of the study, whether it  
22 is cross sectional or prospective and how potential bias  
23 has been adjusted for or avoided and factors like that.

24 Q. Did you employ a method of numerically weighting



1 or scoring the pieces of evidence you evaluated?

2 A. No. I decided not to simply because there are  
3 several adverse effects. The numbers of studies does  
4 not justify an attempt to use particular numbers.

5 Q. Was the weighting of the evidence a product of  
6 your personal judgment?

7 A. It is always a matter of a judgment. I would say  
8 that it is not my personal judgment, but it is a  
9 judgment that would be similar to and I can see that  
10 from ATSDR's report and up to its most recent report,  
11 that it is, we are judging the weight similarly.

12 Q. Do you set out in your report how much weight you  
13 assign to each piece of evidence?

14 MR. WHITLOCK: Objection to the form.

15 A. Yes. I believe so.

16 Q. Where?

17 A. In the section where I, well, in general terms in  
18 the section that I just talked to you about; but then  
19 when we go onto the, my description of (inaudible) at  
20 individual end points, from Page 27 and onwards, that  
21 whole section 8.

22 Q. On Page 27 of your report, you write, I have  
23 cited the most relevant studies. Do you see that?

24 A. I see that.

1 Q. How did you decide what the most relevant studies  
2 are?

3 A. This is a matter of the weight of the study  
4 because I -- no matter what the conclusion was, whether  
5 it contributed to weighty evidence to the basis of, I  
6 mean, the scientific base for making an evaluation of  
7 the evidence.

8 Q. When you say weighty, that as you apply it, in  
9 terms of your report, is a qualitative rather than a  
10 quantitative assessment; is that correct?

11 A. It is not quantitative, because as I said I am  
12 not assigning a number to it but it is a relative  
13 weight.

14 Q. What types of evidence did you weigh in reaching  
15 your opinion?

16 A. I mainly relied on epidemiology, but I did take  
17 toxicology studies into regard without reviewing them in  
18 great detail and in particular I relied on the opinions  
19 summarized by regulatory agencies.

20 Q. What differing weights did you apply to  
21 epidemiological studies as opposed to toxicological  
22 studies?

23 A. Because exactly what we talked about before.  
24 Namely, that could be species differences. I weighed

1 the epidemiology evidence primarily and used the  
2 toxicology information in regard to biological  
3 plausibility.

4 Q. Did you evaluate each piece of evidence on its  
5 own to determine the weight to assign it?

6 A. I did.

7 Q. Was every piece of evidence that you weighed of  
8 the same quality?

9 A. No.

10 Q. Did the studies you considered vary in quality?

11 A. Yes.

12 Q. Did they vary in their limitations?

13 A. They did.

14 Q. Is it fair to say that a study's statistical  
15 method is important for determining the internal and  
16 external validity of that study's findings?

17 A. That is part of it.

18 Q. Did every study that you weighed use the same  
19 statistical method?

20 MR. WHITLOCK: Objection to form.

21 A. I mean, you have to use different statistical  
22 methods, but, of course, it is all a matter of  
23 probability testing, and you could say that is the same  
24 methodology but certainly there are specific methods

1       that vary from study to study.

2           Q. Did you evaluate the statistical method used by  
3 each study before including it in your analysis?

4           A. I did.

5           Q. What was your method for weighting studies  
6 according to the statistical method that they used?

7           A. I don't understand the question.

8           Q. If you applied different weights to each study,  
9 according to the statistical methods, how did you  
10 determine what weight to apply?

11          A. It was not the only aspect. Clearly, if a faulty  
12 statistical method was used for the case, I would take  
13 that into consideration; but your question is so general  
14 it is hard to answer it.

15          Q. Do you agree -- strike that.

16                   Is it your position that in order to be  
17 convinced there is an actual hazard, one needs to have  
18 statistical significance?

19          A. In most cases I would agree. I would also  
20 emphasize that even if something is slightly different  
21 from traditional statistical significance, we still need  
22 to consider it as part of the database.

23          Q. Did you evaluate the statistical significance of  
24 each study before including it in your analysis?

1       A. What do you mean evaluate the statistical  
2       significance, do you mean the methodology or the methods  
3       in general, what do you mean?

4       Q. I mean the findings. Whether the findings were  
5       statistically significant.

6       A. It is part of my evaluation as I said before.

7               MR. WHITLOCK: Objection. Asked and  
8       answered.

9       Q. If a study reported no significant association  
10      between PFOA exposure and an endpoint while another  
11      study reported a significant association, would you  
12      weight both studies equally?

13      A. No.

14      Q. Why not?

15      A. If there is more to take into account, for  
16      example, the size of the study so you can't put that --  
17      that is a misleading question. I am sorry.

18      Q. What was your method for weighing  
19      studies according to whether they reported statistically  
20      significant associations?

21      A. It is part of the weight. As I said to you  
22      before, this is qualitative. It is on a relative scale.  
23      So it is part of the evaluation.

24      Q. Relative risk is one measure of the strength for

1 a statistical association; correct?

2 A. Run that by me again.

3 Q. Relative risk is one measure of the strength for  
4 a statistical association; true?

5 A. I would not say it that way, because clearly you  
6 can have statistical significance with a small relative  
7 risk if the study is large; but you may have a large  
8 relative risk that is not significant if the study is  
9 small.

10 Q. Did you evaluate the relative risk or other  
11 measures of statistical strength when assigning weight  
12 to the evidence that you considered?

13 MR. WHITLOCK: Objection to the form.

14 A. Again, you are trying to put me down on the size  
15 of a relative risk. That is part of it. It is  
16 statistical significance or rather the P value and it is  
17 a magnitude of the relative risk. It all is part of  
18 that evaluation.

19 Q. How did you decide how much weight to assign?

20 A. I think we have been through that because it has  
21 to do with the methodology used, the time span covered,  
22 the size of the population, the methods used. Also, the  
23 methods for ascertaining the outcome. So it is a whole  
24 range of parameters. You cannot put the question down

1 to that simple matter.

2 Q. As I understand it, you took a look at those  
3 factors and perhaps others and then sort of considered  
4 them, and I am not saying this pejoratively, but  
5 cogitated on them for a while and then came up with a  
6 qualitative assessment of how much weight to apply?

7 A. I would not express it that way. I think you  
8 understand it. I would say I agree.

9 Q. Did you consider evidence that reported findings  
10 that were inconsistent with your opinion?

11 A. I did.

12 Q. How did you weight those studies?

13 A. The same way all studies were weighted the same  
14 way.

15 Q. But they did not receive equal weight?

16 A. No.

17 Q. What factors did you decide to apply greater  
18 weight to one study than another study when those  
19 studies had findings that were inconsistent with your  
20 opinions?

21 MR. WHITLOCK: Objection. Asked and  
22 answered.

23 A. Let me just say. When we talk about opinion  
24 here, it is not a matter of personal opinion, but it's

1 an opinion, a conclusion as expressed in this report.  
2 So it is a conclusion rather than, so the word opinion  
3 is not misunderstood. Obviously, one would weigh those  
4 studies equally no matter what the conclusions by the  
5 authors, but I consider that in light of whatever else  
6 is available in that field, the knowledge, the evidence  
7 is available and then I would weigh the different  
8 contributions and then emphasize those that were the  
9 strongest.

10 Q. Again, I don't mean this to be pejorative, but  
11 the only way to, for somebody to audit independently how  
12 you weighed the evidence would be to get inside your  
13 head, true?

14 A. That is not true, because the way that I have  
15 evaluated the evidence here is very similar to like we  
16 talked about before, what IARC has done, WHO, EFSA, EPA  
17 ATSDR, so, I am very similar in my opinion. So you  
18 could say the audit is what groups of colleagues have  
19 decided when working, and I have worked with those  
20 agencies myself in the past, so it is a matter of, you  
21 could say, consensus.

22 Q. On Page 14 of your report, in the middle of the  
23 paragraph below Table 1, you write that it is always  
24 possible for someone critical of the weight of the



1 evidence to raise some type of doubt seeking to require  
2 additional or improved data before a conclusion can be  
3 drawn. Do you see that?

4 A. Yes.

5 Q. Isn't it also possible for someone to use weight  
6 of the evidence to over endorse an opinion?

7 A. Theoretically, yes.

8 Q. On Page 22 of your report, you write, that Doctor  
9 Guzelian refers to the criteria for use of scientific  
10 data in evidence based medicine as applied in regard to  
11 proof of therapeutic effects of drugs for beneficial  
12 human use and that his mistake is to apply the same  
13 criteria in evidence based toxicology regarding proof of  
14 harmful effects. Do you see that?

15 A. Yes.

16 Q. Why in your opinion is that a mistake?

17 A. Because the evidence will never be the same. It  
18 would not have the same strength. With drugs, you can  
19 conduct clinical control trials, which is the highest  
20 level of evidence that we can achieve, and we cannot get  
21 that level of evidence from looking at a toxic substance  
22 like PFOA simply because it is, like we talked about  
23 before, it is both unethical and illegal to those people  
24 with PFOA, as a known toxic substance. While we can do

1 that in regard to drugs where we believe there is a  
2 beneficial outcome that may be achieved by using that  
3 particular drug. That is certainly not the case for  
4 PFOA.

5 Q. Isn't if the method by which one assesses the  
6 results of randomized clinical trials, and assesses the  
7 results of observational epidemiological studies,  
8 essentially the same?

9 A. They are not the same, because in the clinical  
10 control trial, you have a well defined population. You  
11 may even have controls, and blinded controls and you  
12 know exactly what the dose is, because you are  
13 dispensing it yourself. In regard to environmental  
14 toxicants, like PFOA, the dosage is something that can  
15 be observed afterwards. And that means that we are less  
16 certain what the exact dosage was to those subjects.  
17 That very population we talked about before.

18 Q. In your opinion, should the standard for  
19 scientific proof of causation be lower to achieve the  
20 goal of preventing exposure to chemical substance?

21 A. My opinion is that we have to achieve the highest  
22 level of proof possible. That is my opinion.

23 Q. The highest level of proof possible for what?

24 A. For observational studies, in this case with

1 PFOA, and then we have to judge that prudently in order  
2 to reach a respectable decision on what would we allow  
3 our fellow beings to be exposed to.

4 Q. When you say we have to judge that prudently,  
5 what do you mean by prudently?

6 A. Well, exactly what I said. That you will always  
7 be able to raise doubt if you want. Some people do that  
8 because they may have a vested interest. You still have  
9 to reach a decision, I mean, at least that is what the  
10 regulatory agencies have, but can be, and they use the  
11 word tolerated. Not accepted, but tolerated.

12 Q. Are you done with your answer?

13 A. I thought so.

14 Q. I want to be sure. I don't want to speak over  
15 you.

16 In your opinion, is it appropriate to lower  
17 the standard of scientific proof of causation when  
18 exposure to a chemical has already occurred and the only  
19 question is what effects if any will result from that  
20 exposure?

21 MR. WHITLOCK: Objection to the form.

22 A. I think it is a very general question that is  
23 hard to understand. It depends on the circumstances.

24 Q. How about in the circumstances of this case?

1 A. Can you the rephrase the question?

2 Q. In your opinion, is it appropriate to lower the  
3 standard of scientific proof of causation when exposure  
4 to a chemical has already occurred and the only question  
5 is what affects, if any, will result from that exposure?

6 A. I think it is a very theoretical question. I  
7 don't think that has ever happened. Not in my mind.

8 MR. WOLFF: Let's go off the record.

9 THE VIDEOGRAPHER: The time is 11:42. Off  
10 the record.

11 (Whereupon a break was taken)

12 THE VIDEOGRAPHER: We are back on the  
13 record. The time is 11:53.

14 Q. The plaintiffs in this case limit their  
15 allegations to exposure to PFOA; is that correct?

16 A. That is what I understand.

17 Q. Your report's discussion of the epidemiological  
18 and toxicological evidence concerning exposure to  
19 perfluproalkyl substances was not limited to PFOA, was  
20 it?

21 A. It is focused on PFOA. I take into account  
22 relevant evidence on the related substances.

23 Q. For example, your report also refers to PFOS,  
24 PFHXS and PFNA; is that correct?

1 A. Correct.

2 Q. Do you agree the biological response to chemical  
3 substances may be different even though the two  
4 chemicals are part of the same chemical class?

5 A. It could be.

6 Q. Do you know if that is the situation with respect  
7 to the perfluoralkyl substances?

8 A. This is not known in detail yet. The state of  
9 Vermont has decided to look at the total concentration  
10 of PFOA and the related substance in regard to the water  
11 limit.

12 Q. Regardless of what the state of Vermont has done  
13 as a matter of regulatory public health policy, as a  
14 matter of scientific evidence are the biological  
15 responses to PFOA identical to those for PFOS, PFHX,  
16 HSXS, and PFNA?

17 A. In some respects, they appear to be.

18 Q. Are they in all respects?

19 A. I don't think so; but it is a judgment call  
20 because we don't have all of that comparative evidence  
21 to be able to draw conclusions.

22 Q. And in fact on Page 13 of your report, you write  
23 that in studies of exposed communities, exposures are  
24 usually mixed and it may be difficult to distinguish

1 between effects attributable to particular PFASs; is  
2 that correct?

3 A. That's correct.

4 Q. Do you assign different weights to studies that  
5 reported associations involving PFAS substances other  
6 than PFOA?

7 A. I believe just about all of them look at both  
8 PFOA, PFOS, and some of these studies also looked at  
9 others, but those are the two major PFASES.

10 Q. Among other ailments, people have been  
11 experiencing kidney cancer, testicular cancer, bladder  
12 cancer, prostate cancer, steatohepatitis, ulcerative  
13 colitis, osteoarthritis, gout, pregnancy induced  
14 hypertension and asthma for hundreds of years and long  
15 before PFOA was ever synthesized; isn't that correct?

16 A. You are talking about the occurrence. You are  
17 not talking about the risk. So this is correct.

18 Q. On Page 14 of your report in the second  
19 paragraph, you write in the present report, while  
20 considering the extent of possible biases, my  
21 conclusions are stated in terms of assessing elevated  
22 exposure to PFOA results in increased risk of injury or  
23 illnesses. In many instances, the existing evidence of  
24 hazard is much stronger than that, but I understand this

1 to be applicable legal standard; is that correct?

2 A. That is what it says here.

3 Q. You are not a lawyer, are you?

4 A. No.

5 Q. What is your understanding of the applicable  
6 legal standard?

7 MR. WHITLOCK: Objection. Calls for a legal  
8 conclusion. You may answer.

9 A. Again, my civil servant understanding is that it  
10 is more likely than not that PFOA is associated with the  
11 particular outcomes of interest.

12 Q. What is your understanding of what the applicable  
13 legal standard is based on?

14 A. It is not really relevant here, because, I am,  
15 what I am doing is to assess the weight of the evidence  
16 based on the information that is available, and then it  
17 is up to the court to figure out whether, whatever the  
18 legal standard is, whether what I concluded here is  
19 appropriate.

20 Q. Do you know if there is a difference between the  
21 legal standard for regulatory rulemaking and the  
22 imposition of liability in tort?

23 A. I would believe there would be.

24 Q. Do you subscribe to the principle that generally

1 researchers should be conservative when it comes to  
2 assessing causal relationships?

3 A. I believe that researchers should be honest and  
4 apply the weight of the evidence to reach reliable and  
5 prudent conclusions.

6 Q. Is it fair to say that in assessing causation,  
7 researchers first look for alternative explanations for  
8 the association, such as chance, bias or confounding?

9 A. By confounding in my view is part of the bias.  
10 So, yes. That is part of the assessment of the weight  
11 of evidence.

12 Q. In Doctor Ducatman's report, he states that no  
13 one has been declared to have a PFOA associated  
14 condition by any formal body. Do you agree with that  
15 statement?

16 A. You are talking about an individual. That is  
17 correct, because risk is something that operates on  
18 populations. It is a probability.

19 Q. Would you describe the quality of epidemiological  
20 evidence between cigarette smoking and lung cancer to be  
21 irrefutable?

22 A. I would say so.

23 Q. Based on the published data, what is the relative  
24 risk between cigarette smoking and lung cancer?



1           A. I don't know. I don't remember that by heart.

2           Q. How would you describe the quality of  
3 epidemiological evidence between asbestos exposure and  
4 mesothelioma?

5           A. I would say that it is much stronger.

6           Q. Based upon the published data, what is the  
7 relative risk between asbestos exposure and  
8 mesothelioma?

9           A. How was that different from the question before?

10          Q. One was asking you to assess the quality and the  
11 other is for a relative risk.

12          A. A relative risk. So an elevated relative risk,  
13 are you asking me --

14          Q. Let me ask you again.

15                   Based upon the published data, what is the  
16 relative risk between asbestos and mesothelioma?

17          A. It is highly elevated and highly significant, but  
18 I don't remember the exact magnitude.

19          Q. When you say highly elevated, can you give me a  
20 number and a range, a ballpark range that you're  
21 thinking of?

22          A. No. Not by heart.

23          Q. Can you compare and contrast the quality of the  
24 epidemiological evidence between cigarette smoking and

1 lung cancer with the body of data on PFOA exposure and  
2 kidney cancer?

3 A. Which way do you want me to do that?

4 Q. In any way you feel comfortable.

5 A. Well, with regard to kidney cancer, we have to  
6 say that there are much fewer studies. Therefore, the  
7 weight of the evidence is less than it is in regard to  
8 tobacco smoking and lung cancer.

9 Q. Compare and contrast the quality of the  
10 epidemiological evidence between cigarette smoking and  
11 lung cancer with the body of data on PFOA exposure and  
12 testicular cancer?

13 A. My answer is the same. There are much fewer  
14 studies. Therefore, the evidence is less solid.

15 Q. Compare and contrast the quality of the  
16 epidemiological evidence between cigarette smoking and  
17 lung cancer with the body of data on PFOA exposure and  
18 prostate cancer?

19 A. I would say that the quality of the studies are  
20 generally not different. It is just a number of studies  
21 that have been conducted.

22 Q. Compare and contrast the quality of the  
23 epidemiological evidence between cigarette smoking and  
24 lung cancer with the body of data on PFOA exposure and

1     steatohepatitis?

2           A.   My answer is the same.

3           Q.   Compare and contrast the quality of  
4     epidemiological evidence between cigarette smoking and  
5     lung cancer with the body of data on PFOA exposure and  
6     osteoarthritis?

7           A.   My answer is the same.   Much less evidence on  
8     that.

9           Q.   Throughout your report, you refer to the C8  
10    Health Project; is that correct?

11          A.   That's correct.

12          Q.   On Page 18 of your report in the third full  
13    paragraph, you state that these data and the conclusions  
14    released by the science panel constitute an important  
15    basis for the present report.   Based on the results from  
16    these studies and an evaluation of the literature, the  
17    science panel delivered on, quote, probable links, close  
18    quote as summarized in the file report from 2012.   True?

19          A.   Correct.

20          Q.   At the top of Page 18 of your report, you note  
21    that as a result of a settlement with Dupont, the  
22    settlement agreement created a science panel of three  
23    epidemiologists that was to conduct research in the  
24    community to evaluate probable links between PFOA

1 exposure and human disease; is that correct?

2 A. That's correct.

3 Q. Are you aware that by a negotiated agreed upon  
4 settlement, Dupont has been funding a C8 Health Project  
5 and a medical monitoring program?

6 A. I don't remember the details; but I think this is  
7 correct.

8 Q. On Page 18 of your report in the third paragraph,  
9 you state that based on the results from these studies  
10 and an evaluation of the literature, the science panel  
11 delivered reports on, quote, probable links, close  
12 quote, as summarized in the report from 2012, correct?

13 A. This is what you read before. That's correct.

14 Q. Why did you put the phrase probable links in  
15 quotation marks?

16 A. Because that was a wording that was chosen  
17 between the experts and the court as far as I remember.  
18 Therefore, it had a specific meaning in this connection.

19 Q. Do you know what that specific meaning was?

20 A. I don't remember it, but it was something that,  
21 the meaning was in order to satisfy both the court and  
22 the scientists.

23 Q. In order to satisfy the scientists in connection  
24 with the way that they would normally go about assessing

1 causal relationships or something else?

2 A. I think that they assess the causal relationships  
3 and the weight of the evidence the same way they would  
4 have done otherwise, but the court clarified somehow to  
5 them that they should not try to aim for some more or  
6 less perfect proof, but that a probable link meaning  
7 that there was a clearly greater risk associated with  
8 particular outcomes at elevated exposures to these  
9 substances. That's my understanding.

10 Q. Is it the same standards that the epidemiologists  
11 would apply in their academic work?

12 A. It may depend. I can't vouch for all  
13 epidemiologists but I can say that in general I would  
14 think that we would aim for stronger evidence of causal  
15 relationships and just a probable link.

16 Q. In which scenario, in which context?

17 A. In the scientific context. We try to achieve the  
18 best evidence possible.

19 Q. Did you put the phrase probable links in  
20 quotation marks because it is not a generally accepted  
21 standard in epidemiology or clinical medicine?

22 MR. WHITLOCK: Objection to the form.

23 A. I don't have any knowledge of that, but the  
24 reason that I did it was simply to indicate that it had

1 a specific meaning. And if I had not put it in  
2 quotation marks, people would say, oh, that's just  
3 probable links and whatever popular understanding. To  
4 indicate that this was a wording that had been agreed  
5 upon with the court.

6 Q. So you are aware that a so called probable link  
7 is an agreed to legal standard and not a scientific  
8 standard between PFOA exposure and certain health  
9 conditions?

10 A. I think it is both. It was something that was  
11 agreed with the three scientists; but of course I was  
12 not part of that discussion. So I cannot be sure.

13 Q. Were you aware that during its review, the C8  
14 Science Panel was repeatedly reminded by the plaintiffs'  
15 counsel not to imply typical scientific standards or  
16 methods used to determine causation but rather something  
17 quite different?

18 A. I have no insight into this.

19 MR. WOLFF: Mark this as Exhibit Number 5.

20 (Letter dated January 22, 2010 was marked as  
21 Exhibit Number 5 for identification)

22 Q. Exhibit Number 5 is a January 22, 2010 letter  
23 from the plaintiffs' counsel in the Dupont litigation,  
24 Robert Hillot to the C8 Science Panel. Have you

1 previously seen this letter?

2 A. I don't think so.

3 Q. Please turn with me to Page 5. That is the last  
4 page. The last paragraph. Were you aware that the  
5 plaintiffs' counsel admonished the panel over the  
6 distinction between the probable link standard and the  
7 inapplicable causation standard that the panel may be  
8 more familiar with from its prior academic and  
9 scientific work?

10 A. I can see that.

11 Q. Were you aware that the plaintiffs' counsel told  
12 the science panel to keep that distinction in mind?

13 A. No. That is not relevant to my opinion.

14 Q. Please, turn with me to Page 3 in the first full  
15 paragraph. Were you aware that the plaintiffs' counsel  
16 emphasized to the C8 Science Panel that the distinction  
17 between the causation standard and the more lenient,  
18 quote, probable link, close quote, standard of proof is  
19 of utmost importance in that it is a fundamental  
20 distinction in the level and quantity of evidence  
21 necessary to make the determination at issue. Were you  
22 aware of that?

23 A. No. I can understand what it says here. I think  
24 my understanding that we discussed before is correct.

1 Q. Please turn with me to Page 4 at the bottom.

2 Were you aware that the C8 Science Panel was instructed  
3 by the plaintiffs' counsel that if there just enough  
4 evidence to tip the scales towards a finding of any link  
5 between PFOA exposure and any human disease even in the  
6 slightest degree, then the probable link standard has  
7 been met. Were you aware of that?

8 A. No. Again, it is not relevant to my opinion.

9 Q. Why do you say it is not relevant to your  
10 opinion?

11 A. Because we are now in 2018. Most of the evidence  
12 that was collected by the three scientists has been  
13 published in peer review journals.

14 MR. WOLFF: Mark this as Exhibit Number 6.

15 (Letter dated August 2, 2015 was marked as  
16 Exhibit Number 6 for identification)

17 Q. Exhibit Number 6 is an August 2, 2005 letter from  
18 the plaintiffs' counsel in the Dupont C8 litigation.  
19 Larry Winter, to the C8 Science Panel. Have you  
20 previously seen this letter?

21 A. I don't believe so.

22 Q. Please turn with me to Page 3. The last  
23 paragraph. Were you aware that at least as early as  
24 August of 2005, the plaintiffs' counsel told the C8



1 Science Panel members that we recognize that the  
2 imposition of a legal standard on scientific analysis  
3 presents various problems and we appreciate that the  
4 science panel is accustomed to employing proof  
5 requirements that are no doubt more stringent than those  
6 that govern and control your work here. You have each  
7 confirmed that you can follow the unique, quote,  
8 probable link, close quote, standard required in this  
9 matter regardless of how inconsistent that standard may  
10 be with traditional causation analysis. Were you aware  
11 that the science panel was told that?

12 A. I think the general understanding that I  
13 described before remains correct. I was not aware of  
14 this particular wording, but I don't think this changes  
15 anything in what I just said before.

16 Q. Were you aware that two members of the C8 science  
17 panel subsequently testified under oath in 2011 that  
18 their, quote, probable link, close quote, reports are  
19 not susceptible to scientific peer review and would not  
20 be submitted for publication and peer reviewed  
21 scientific journals?

22 MR. WHITLOCK: Objection to form. Lack of  
23 foundation.

24 A. Well, this contrasts to my understanding, is that

1 most of what this evidence is about in regards to this  
2 C8 panel has actually been submitted to peer review  
3 journals and published. This is what I am relying on.  
4 I am not simply relying on the probable links reports.

5 Q. Isn't it fair to say the so called probable link  
6 findings do not establish causation between PFOA  
7 exposure and any adverse health effects in humans?

8 A. Again, what you are asking me is to repeat myself  
9 that I have the understanding of what the probable links  
10 are all about, and that it is something that has been  
11 negotiated with the court. So I was not part of that.  
12 So I have not taken that into consideration. Nor have I  
13 considered these letters.

14 Q. We are done with those letters.

15 Do you agree that attributing causation to a  
16 particular chemical exposure in a specific instance is a  
17 challenge when there has been exposure to multiple  
18 chemicals?

19 A. It is always a challenge.

20 Q. In the real world, everyone is exposed to many  
21 chemicals on any given day; is that correct?

22 A. Yes, but you have to consider the order of  
23 magnitude of those exposures.

24 Q. Those exposures can be multiplied over a

1       lifetime?

2           A.   I would not use the word multiply, but the number  
3       would be of course larger.

4           Q.   What word would you use?

5           A.   I would just say the number would be greater.

6           Q.   Is it fair to say though some groups of people  
7       are exposed to different combinations of chemicals in  
8       different doses?

9           A.   That's true.

10          Q.   In your report, you cite several of your own  
11       research articles reporting associations between PFAS  
12       exposure and reduced antibody concentrations, increased  
13       body mass, asthma, and shortened duration of breast  
14       feeding based on studies in the Faroe Islands?

15          A.   Yes.

16          Q.   The Faroe Islands are a remote archipelago in the  
17       North Atlantic, correct?

18          A.   Some people might say they are remote. They are  
19       in between Norway and Iceland.

20          Q.   The Faroese diet is traditionally high in whale  
21       meat and whale blubber?

22          A.   I would say it used to be.

23          Q.   As of when?

24          A.   There have been advisories issued by the

1 government or the medical director of the Faroe Islands  
2 to limit the populations use of whale meat and whale  
3 blubber. I think the first one was issued, I try to  
4 recall, 1998.

5 Q. Notwithstanding those advisories, the Faroese  
6 diet continues to be characterized by the consumption of  
7 whale meat and whale blubber among other things?

8 A. That is not correct. It is a population that's  
9 characterized by marine food. When I say marine, it is  
10 primarily fish. That is species of fish also eaten in  
11 the US, and shellfish, and comparatively speaking, the  
12 average daily intake at the peak was something like  
13 fourteen grams of whale meat per day for adults. So I  
14 can't say this was a major contribution to the diet.

15 Q. The marine based diet of the Faroese population  
16 exposes them to a number of environmental chemicals; is  
17 that correct?

18 A. Yes.

19 Q. Those chemicals exposures include methylmercury?

20 A. Methylmercury occurs in the meat of whales just  
21 like in tuna and other similar species eaten in the  
22 United States.

23 Q. The chemical exposures of the marine based diet  
24 include PCBs, is that correct?

1 A. Yes. PCBs occur in the blubber.

2 Q. The marine based diet confers exposures to DDT;  
3 is that correct?

4 A. DDT often occurs along with PCBs in marine food.

5 Q. The marine based diet confers exposures to DDE,  
6 correct?

7 A. DDE is a metabolic of DDT.

8 Q. And the marine based diet confers exposures to  
9 trans-nonachlor; is that correct?

10 A. I don't believe so. And, yes, it is a very small  
11 contribution.

12 MR. WOLFF: Mark this as Exhibit Number 7.

13 (Chapter on Human Health in the Faroe  
14 Islands was marked as Exhibit Number 7 for  
15 identification)

16 Q. Exhibit Number 7 is a chapter on human health in  
17 the Faroe Islands from a national report available from  
18 the ministry of environment and food of Denmark. Have  
19 you seen this publication before?

20 A. I'm sorry. I can't remember. I must have seen  
21 it.

22 Q. Help me with the pronunciation, the first named  
23 authority? Doctor Weihe, the lead author of this chapter  
24 is someone that you have frequently collaborated with in

1 your studies of the Faroese; is that correct?

2 A. That's correct.

3 Q. On Page 177, in the last paragraph, the  
4 investigators write that the amount of whale meat  
5 consumed during pregnancy is the best predictor for  
6 maternal methylmercury exposure; is that correct?

7 A. Yes.

8 Q. And continuing, they write that any form of  
9 blubber, dried or fresh, seems to be the major source  
10 not only for PCB but also for DDT, DDE, and  
11 trans-nonachlor.

12 A. All right.

13 Q. Those last three chemicals had applications as  
14 pesticides; is that correct?

15 A. DDT did. DDE is a metabolite so it wasn't used  
16 by itself. But it's a metabolic tran-nonachlor was used  
17 as I understand.

18 Q. As a pesticide?

19 A. Yes. It is all history.

20 Q. The report also states that the Faroese exposure  
21 to these chemicals is also associated with the  
22 consumption of fulmar, a diet of sea mammals, fatty fish  
23 and perhaps eggs from seabirds and Puffin; is that  
24 correct?

1           A.   That's what it says.

2           Q.   Whale meat and blubber, seabirds and their eggs  
3   and Puffin are not typical components of the average  
4   diet in Vermont or New Hampshire, are they?

5           A.   I think this is -- your statement is correct.  
6   This is a different diet.

7                     MR. WOLFF:   Mark this as Exhibit Number 8.

8                     (Study was marked as Exhibit Number 8 for  
9   identification)

10          Q.   Exhibit Number 8 is a study entitled Cohort  
11   Studies of Faroese Children Concerning Potential Adverse  
12   Health effects after mother's exposure to marine  
13   contaminants during pregnancy; is that correct?

14          A.   Correct.

15          Q.   And the only two co-authors on this study are  
16   Doctor Weihe and you; is that correct?

17          A.   That's correct.

18          Q.   On Page 4 in the first bolded heading, you wrote  
19   the -- strike that.

20                     On Page 4, the first bold heading you quote  
21   the contaminants of the blubber adversely affect the  
22   immune system so that the children react more poorly to  
23   immunizations; is that correct?

24          A.   That's correct.

1 MR. WOLFF: Mark this as Exhibit Number 9.

2 (Paper was marked as Exhibit Number 9 for  
3 identification)

4 Q. Exhibit Number 9 is a paper on which you are a  
5 co-author. First author is Kileman from 2010. Serum  
6 concentrations of antibodies against vaccine toxoids in  
7 children; is that correct?

8 A. That's correct.

9 Q. On Page 1434, the bottom of the middle column,  
10 you wrote that the Faroe Islands provide a unique  
11 opportunity to study PCB immunotoxicity given the PCP  
12 contamination of pilot whale blubber, a traditional food  
13 item favored by many Faroese, correct?

14 A. But not all.

15 Q. That's what you wrote?

16 A. Yes.

17 MR. WHITLOCK: But not all.

18 A. But not all.

19 Q. You concluded that developmental PCB exposure is  
20 associated with immunotoxic effect on serum  
21 concentrations of specific antibodies against diphtheria  
22 and tetanus vaccinations. The immune system  
23 development during the first years of life appears to be  
24 particularly vulnerable to this exposure. That's what



1     you wrote; is that correct?

2           A.   That's correct.

3           Q.   And that is essentially the same hypothesis that  
4     you are advancing in this case except that here it  
5     concerns PFOA; is that correct?

6           A.   I need to explain this, because when we wrote  
7     this paper, we had not yet measured the PFAS  
8     concentrations. So this is based alone on PCBs and we  
9     have not taken into regard the potential confounding  
10    from PFOA.

11          Q.   On Page 29 of your report, Paragraph C, you  
12    describe reported findings from a Faroese birth cohort  
13    for concentrations of antibodies against antibodies and  
14    diphtheria.

15          A.   Point me to it?

16          Q.   Paragraph C.

17          A.   Okay. Can you repeat for me the question.

18          Q.   Well, you described a study that reported  
19    findings from a Faroese birth cohort for concentrations  
20    of antibodies against tetanus and diphtheria. Do you see  
21    that?

22          A.   Yes.

23          Q.   You don't provide a reference or end note for  
24    that study, do you?

1       A. It may not have, well, it is elsewhere in the  
2 report. It may not have been published at the time but  
3 it has been recently published. Before August 1, it was  
4 published.

5               MR. WOLFF: Mark this as Exhibit Number 10.

6               (Study was marked as Exhibit Number 10 for  
7 identification)

8       Q. Exhibit Number 10, is this the study that you are  
9 referring to?

10      A. Yes. That is the one.

11      Q. Good. In your report at Page 29, you write that  
12 the association support the notion that the developing  
13 adaptive immune system is particularly vulnerable to  
14 immunotoxic exposures to for example PFOA during  
15 infancy?

16      A. Yes.

17      Q. If you substitute PCBs for PFOAs as the example,  
18 and the notion that developmental exposure to PCBs can  
19 reduce vaccine responses in children, is essentially the  
20 same hypothesis that you are advancing with respect to  
21 PFOA here?

22      A. That's fine. There is no problem in having one  
23 compound having the same or overlapping outcomes as  
24 another one. So I don't see a problem.

1 Q. The Faroese marine diet is the source of the  
2 exposure to these chemicals; is that correct?

3 MR. WHITLOCK: Objection to the form.  
4 Vague.

5 A. With PFOA, if I may focus on PFOA the association  
6 with marine diet is not so strong. It probably comes  
7 from other consumer products and for example food  
8 wrapping materials because it does not occur in drinking  
9 water.

10 Q. So if there is no PFOA in the drinking water in  
11 the Faroe Islands, and the marine based diet is not the  
12 source of the PFOA exposure, how are the Faroese being  
13 exposed to PFOA?

14 A. Right. They expose exactly the same way as  
15 American, and Danes, and other Europeans in that PFOA is  
16 used for multiple purposes in society and the  
17 concentrations that the Faroese have are similar to what  
18 Americans have.

19 Q. In your 2010 study, you reported that decreased  
20 serum concentration of antibodies were attributable to  
21 PCB exposure, correct?

22 A. I need to make sure that you are talking about  
23 Exhibit Number 9.

24 Q. I am.

1       A.   Okay.   So I stand by that.   I also said we did  
2       not measure PFAS at this time.   When we found out that  
3       PFAS had the same affect and was much stronger, then we  
4       adjusted the PFAS association, that is, PFOA and the  
5       related compounds for PCB exposure.   That is what is in  
6       my 2012 article in JAMA.

7       Q.   In your 2010 study, Exhibit Number 9, on Page  
8       1438 in the middle column, you wrote, the Faroese  
9       population is also exposed to contaminants other than  
10      PCBs such as PPDDE, hexachlorobenzene and beta  
11      hexachlorocyclohexane, because they correlate with PCB,  
12      this study could not assess whether they played any  
13      independent role; is that what you wrote?

14      A.   That's correct.

15      Q.   Exposure to PCB, mercury, and other chemicals is  
16      a potential covariate and confounder of any reported  
17      association between PFOA and a health-related endpoint  
18      in your study on the Faroese; is that correct?

19      A.   Where do you have that wording from?

20      Q.   I am asking a question.

21      A.   This is your wording?

22      Q.   Yes.

23      A.   Let me then say that PCB would be a confounder  
24      based on the degree of association with PFOA, because if

1     there is no association, you will not have any  
2     conformity.

3           Q. Did all of your studies on PFAS exposure on the  
4     Faroese control for exposure to multiple PCB congeners?

5           A. I think we narrowed this down to total PCB. We  
6     did the calculations, also, for individual major  
7     congeners and the findings were the system.

8           Q. So it was not across the board?

9           A. You can't, because some of them are not  
10    detectable. So you just have zero exposure. And these  
11    statistics are not very reliable. If there is something  
12    that you cannot measure.

13          Q. Did all of your studies of PFAS exposure of the  
14    Faroese control for exposure to mercury, DDT, DTE, and  
15    hexochlorobenzene?

16          A. Not necessarily. Yes to mercury. We looked at  
17    it. There is no reason to test for confounding if it is  
18    not associated with PFOA exposure, and in this case,  
19    mercury was not associated with the antibodies. With  
20    DDE, it is a little more difficult because it is so  
21    closely associated with PCB, and if PCB is not a  
22    confounder, then the conclusion is that then PDE is not  
23    confounder.

24          Q. Did any of your studies of PFAS exposure on the

1     Faroese control for trans-nonachlor or  
2     beta-hexachlorocyclohexane?

3           A. It is not necessary to do so because we took into  
4     account PCB as the major pollutant because the exposures  
5     to those other substances that you are mentioning,  
6     particularly trans-nonachlor is much, much lower and  
7     highly questionable relevance.

8                   MR. WOLFF: Off the record.

9                   THE VIDEOGRAPHER: The time is 12:36. Off  
10    the record.

11                   (Whereupon a lunch break was taken)

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1 AFTERNOON SESSION

2 WITNESS RESUMED

3 THE VIDEOGRAPHER: Back on the record. The  
4 time is 1:29 p.m.

5 Q. Mr. Whitlock, counsel for the plaintiffs  
6 previously stipulated that the plaintiffs are not  
7 proffering you as an expert in medical monitoring. Do  
8 you recall that?

9 A. Right.

10 Q. Do you agree with that assessment?

11 A. Yes. Because it is in accordance with my  
12 introduction on Page 1.

13 Q. Is that because you are an expert in epidemiology  
14 and the substance and analysis in your report is  
15 addressed to epidemiology as opposed to medical  
16 monitoring?

17 A. No. It was just my understanding that what I  
18 have done here is what was needed and I was asked to  
19 challenge rebuttals from the three defense experts.

20 Q. So rather than addressing a medical monitoring  
21 program, your report speaks to epidemiological issues;  
22 is that correct?

23 A. And the need for medical monitoring. It  
24 certainly does but I did not delve into the details of

1 that.

2 Q. Let's return to the Faroe Islands.

3 A. Okay.

4 Q. Can you point to epidemiological evidence that  
5 PCB exposure is associated with an increase in the  
6 number of cases of diptheria in children in the Faroe  
7 Islands?

8 A. I don't think diptheria has occurred in recent  
9 years in the Faroes.

10 Q. Can you point to epidemiological evidence that  
11 PCB exposure is associated with an increase in the  
12 number of cases of tetanus in children in the Faroe  
13 Islands?

14 A. Again, I am not aware of any cases of tetanus in  
15 recent years in the Faroe Islands.

16 Q. Can you point to epidemiological evidence that  
17 PCB exposure is associated with an increase in mortality  
18 from colds or fevers in children in the Faroe Islands?

19 A. There may be such increase but I am not aware it  
20 is associated with PCB exposure.

21 Q. Now, I take it that since you not aware of any  
22 increase in the number of cases of diptheria, you cannot  
23 point to epidemiological evidence that PFOA exposure is  
24 associated with an increase in the number of cases of



1     diphtheria in children; is that correct?

2           A.   That's correct.   If we are just looking at  
3     diphtheria.

4           Q.   Similarly, with respect to tetanus, you cannot  
5     point that epidemiological evidence that PFOA exposure  
6     was associated with an increase of number of cases of  
7     tetanus in children, is that true?

8           A.   True.

9           Q.   Can you point to epidemiological evidence that  
10    PFOA exposure is associated with an increase in  
11    mortality from colds or fevers in children in the Faroe  
12    Islands?

13          A.   There is no such evidence as it has not been  
14    looked for.

15          Q.   Do you agree that antibody responses of normal  
16    children to routine prophylactic vaccinations can vary  
17    substantially?

18          A.   They can vary.   That's correct.

19          Q.   The reasons for this wide variation are poorly  
20    understood; true?

21          A.   That is correct.   It is not greatly understood.

22          Q.   Do you agree that smoking during pregnancy can  
23    have long term adverse health affects on a fetus?

24          A.   Do you mean beyond birth or do you mean during

1       gestation?

2           Q.   Beyond birth.

3           A.   Beyond birth.  I agree, so, yes.

4           Q.   Would you agree that a person's genetic make up  
5       plays a substantial role with regard to health and  
6       longevity and mortality?

7                       MR. WHITLOCK:  Objection to the form.  
8       Vague.

9           A.   This is correct.  Genetic make up may even make a  
10       subject more vulnerable to PFOA but we don't know the  
11       detailed evidence yet.

12          Q.   The Faroe Islands are by definition insular; is  
13       that correct?

14          A.   Yes.  The Faros are a number of islands in the  
15       North Atlantic.

16          Q.   And the Faroese are a fairly homogenous  
17       population; true?

18          A.   Yes.  This is what I have claimed at least.

19          Q.   In your opinion, these factors make the Faroese  
20       an advantageous study population; is that correct?

21          A.   This is what I have written, yes.

22          Q.   Is it fair to say that you believe that this  
23       makes the reported findings on developmental exposures  
24       to be generalizable to other populations beyond the

1 Faroe Islands?

2 A. I believe so. Yes.

3 Q. The Faroese are a fairly genetically homogenous  
4 population; is that true?

5 A. That's what I understand.

6 Q. And an isolated and genetically homogeneous  
7 population can be particularly susceptible to higher  
8 rates of heritable disorders; is that correct?

9 A. Yes. To some degree.

10 Q. And the frequency of these heritable disorders in  
11 the Faroe Islands is unlikely to be similar to that in  
12 Vermont or New Hampshire; is that correct?

13 A. Right.

14 Q. And the Faroese are predisposed to primary  
15 carnitine deficiency?

16 A. Yes. That has been discovered.

17 Q. Carnitine is found in the acidity of fatty acids  
18 and glucemic regulation; true?

19 A. I am not sure how important it is in regards to  
20 glucose metabolism, but certainly with regard to fatty  
21 acid.

22 Q. Do you agree when the body is unable to oxidize  
23 fatty acids, that fats accumulate in the liver, skeletal  
24 muscle and the heart?

1           A. I would have to look it up. I believe this is  
2 correct.

3           Q. Liver steatosis also has an affect on the liver's  
4 failure to properly metabolize fats; is that true?

5           A. I think so.

6           Q. Did your studies of the Faroese control for the  
7 incidents of carnitine deficiencies?

8           A. Yes. To the extent those cases were identified  
9 we did not include them in the study.

10          Q. Are you aware that researchers have reported that  
11 the Faroese have the highest prevalence of glycogen  
12 storage disease type 3A in the world?

13          A. I don't remember. It is likely true.

14          Q. Isn't it true that beginning in infancy,  
15 individuals with any type of glycogen storage disease  
16 type 3 may have low blood sugar, excess amounts of fats  
17 in the blood, and elevated blood levels of liver enzymes  
18 and that as they get older, children with this condition  
19 typically develop an enlarged liver?

20                 MR. WHITLOCK: Objection to the form.

21          A. I think what you are saying is correct. It is  
22 not relevant to my studies.

23          Q. Are you aware of any statement in the peer review  
24 scientific and medical literature which reports PFOA is

1 a cause of kidney cancer?

2 A. Not in the Faroes but in general?

3 Q. In general.

4 A. It is a cause, I think the wording that has been  
5 used is risk factor.

6 Q. And a risk factor is not synonymous with a cause,  
7 is it?

8 A. It depends on the circumstances. Because in  
9 scientific literature, we don't necessarily use the word  
10 causation but use a risk factor as something that we  
11 need to adjust for when we consider potential  
12 confounding.

13 Q. Is cigarette smoking a risk factor for cirrhosis  
14 of the liver?

15 A. I don't remember. It may well be.

16 Q. Does cigarette smoking cause cirrhosis of the  
17 liver?

18 A. Again, I can't give you an answer because it is  
19 not something that I remember.

20 Q. Isn't it true that people who tend to drink large  
21 amounts of alcohol also tend to smoke cigarettes and for  
22 that reason cigarette smoking is a risk factor for  
23 cirrhosis?

24 A. I would not use that wording. I would say that

1 it is a potential confounder.

2 Q. Are you aware of any statement in the peer  
3 review, scientific and medical literature which reports  
4 that purports PFOA is a cause of testicular cancer?

5 A. Again, it may well be written somewhere in the  
6 literature. I do not remember it. I would think the  
7 wording used is a risk factor.

8 Q. Are you aware of any statement in the peer review  
9 scientific and medical literature which reports that  
10 PFOA is a cause of steatohepatitis?

11 A. Again, I don't remember.

12 Q. Are you aware of any statement in the peer review  
13 scientific and medical literature which reports that  
14 PFOA is a cause of any particular disease in humans?

15 A. Again, I can't answer you. I don't remember.

16 Q. You would agree that every individual has his or  
17 her own background risk of developing the diseases that  
18 you describe on Pages 27 through 62 of your report; is  
19 that correct?

20 A. Right. We all have different background  
21 depending on age, and sex, other factors.

22 Q. Hereditary factors can contribute to an  
23 individual's background risk; is that true?

24 A. I agree.

1 Q. And an individual's level of physical activity  
2 can affect their background risk for some health  
3 endpoints; is that correct?

4 A. That's correct.

5 Q. And eating habits affect an individual's  
6 background risk as well, true?

7 A. It depends on the outcome. I would agree in  
8 general for some outcomes, yes.

9 Q. The individual's age at a given point in time is  
10 also a factor when evaluating one's background risk; is  
11 that true?

12 A. I would agree.

13 Q. Please assume with me that there are two  
14 individuals. Individual Number 1 is a forty-year old  
15 male. Never a smoker. Individual Number 2 is a  
16 forty-year old male, a pack a day cigarette smoker since  
17 his teen years. What would you estimate is the  
18 approximate relative risk of non small cell lung cancer  
19 of Individual Number 1 versus Individual Number 2?

20 A. I can't give you that number. I would have to  
21 look it up.

22 Q. Why can't you give that number?

23 A. Well, because I don't remember it. You can have  
24 a small cell lung cancer if you are a non-smoker. Those

1 cases occur. It could perhaps be attributed to indirect  
2 smoking. It could be perhaps effected by occupational  
3 exposure, could be effected by genetic susceptibility.  
4 So that risk, who's non-smoker is not necessarily zero  
5 or close to zero, but certainly everything else being  
6 equal, the smoker would have a higher risk.

7 Q. How would you know that?

8 A. How would I know that? I would rely on the  
9 scientific literature and my judgment.

10 Q. Are you referring to epidemiological studies or  
11 something else?

12 A. No. This is based on epidemiology and I would  
13 say the assessment of IARC.

14 Q. Please assume that a 65 year old man is diagnosed  
15 with non small cell lung cancer and that he has a forty  
16 year pack cigarette smoking history. Besides tobacco  
17 smoking, what are the other possible causes of his lung  
18 cancer?

19 A. Again, I don't remember the details. So I would  
20 have to look it up.

21 Q. Can you give me any possible other causes?

22 A. Not at this point. I have not looked at that  
23 literature lately.

24 Q. Would an unknown cause be a possible cause of his



1 lung cancer?

2 A. Again, I have to make some assumptions. So I  
3 can't really give you a good answer today.

4 Q. What would be the most probable cause of this  
5 individual's lung cancer?

6 A. The forty pack a year -- pack a day smoker.

7 Q. So the sixty-five year old man, forty pack year  
8 smoking history?

9 A. Forty pack years?

10 Q. Yes. Not forty packs a day.

11 A. Okay. You are asking me to make assumptions. I  
12 would rather not answer the question.

13 Q. Well, you understand that as an expert witness, I  
14 can ask you to make assumptions?

15 A. That is fair enough. Let's stay with the topic  
16 that is related to my expert report.

17 Q. Please assume that a 65 year old man who has been  
18 a metal worker for forty years develops COPD. Please  
19 further assume that OSHA and state agencies have  
20 repeatedly cited his employer over the term of his  
21 employment for violating occupational exposure standards  
22 for mineral fumes, welding fumes, and sulfur dioxide,  
23 what would you say are the possible causes of his COPD?

24 A. Again, I have to reserve my judgment. You are

1 giving me personal information and you are saying that  
2 an agency has cited the employer, but I don't know what  
3 the basis of that citation and I don't know what the  
4 levels of exposure are and what, how they are compared  
5 to regulatory guidelines. So I can't answer that  
6 question.

7 Q. Other than the occupational exposure to the  
8 substances mentioned, what are the other possible causes  
9 of his COPD?

10 MR. WHITLOCK: Objection. Calls for  
11 speculation.

12 A. Yes. Certainly smoking is a risk. I am sure  
13 that there is a genetic component as well. I would like  
14 to limit it to that because I have not prepared for  
15 questions like this.

16 Q. Is an unknown cause also a possible cause of the  
17 COPD?

18 A. Unknown causes are always possible until they  
19 have been discovered because then they turn to be known  
20 risk factors.

21 Q. What is the probability that his COPD is due to a  
22 cause other than occupational exposure?

23 A. I would refer to my answer before that I have not  
24 prepared to answer questions like this.

1 Q. Please assume that there are two individuals.  
2 Individual Number 1 is a 25 year old female with a BMI  
3 of 31 living for the past three years in a town where  
4 the PFOA level in the drinking level is 70 parts per  
5 trillion. Individual Number 2, 25 female with a BMI of  
6 31 with no known exposure to PFOA. What would you  
7 estimate is the approximate relative risk of developing  
8 Type 2 diabetes in Number 1 versus Number 2?

9 MR. WHITLOCK: Objection to the form.

10 A. Again, I am looking at this from a population  
11 viewpoint. I am not looking at individual risk. So I  
12 can give you information about epidemiology as we have  
13 looked at female nurses in America and I'm sure some from  
14 Vermont in regard to their BMI and their blood  
15 concentration of PFOA; but I would have to look up that  
16 information because I don't remember the details on 25  
17 years. And BMI of 31. I can't just offhand give you a  
18 number.

19 Q. Please assume that one year later, individual  
20 Number 2, that is the woman with no known exposure to  
21 PFOA develops Type 2 diabetes. What are the possible  
22 causes of her Type 2 diabetes?

23 MR. WHITLOCK: Objection to the form.

24 A. Let me just say that genetic predisposition is a

1 known factor. And when we are talking about  
2 individuals, we can't easily refer to particular  
3 causation, but of course in this case if there is a  
4 family history for Type 2 diabetes, it is obvious that  
5 must somehow have contributed.

6 Q. Are there any other possible causes of her Type 2  
7 diabetes?

8 A. Not that I wish to discuss right now.

9 Q. Is an unknown cause a possible cause of her Type  
10 2 diabetes?

11 A. I refer to my previous response that unknown  
12 causes is always possible until you find out what they  
13 are and then they turn out to be known causes.

14 Q. What is the most probable cause of her Type 2  
15 diabetes?

16 MR. WHITLOCK: Objection to the form.

17 A. This all depends on sex and age. I am not  
18 prepared to give you a complete discourse on that.

19 Q. So here we have a female who is 26 years old.

20 A. What about it?

21 Q. Well, I have just given you the sex and the age.

22 A. Right. What's your question? I don't  
23 understand.

24 Q. What's the most probable cause of her Type 2

1 diabetes? A 26 year old female with a BMI of 31 with no  
2 known exposure to PFOA.

3 MR. WHITLOCK: Objection to the form.

4 A. Given that the BMI is elevated, I think it would  
5 probably had a role in the causation. That's all I can  
6 say at this point.

7 Q. Please, assume that five individuals, let's call  
8 them A, B, C, D, and E, are males, all of the same age,  
9 and BMI, who have lived in a town with the PFOA level in  
10 the drinking water is seventy parts per trillion. In  
11 your opinion, do all five have the same probability of  
12 developing ulcerative colitis or thyroid disease or  
13 kidney cancer or hypercholesterolemia or testicular  
14 cancer?

15 A. From the information given to me, I would say  
16 yes.

17 Q. Why is that?

18 A. Because they have exactly the same  
19 characteristics.

20 Q. Please assume that subject -- strike that.

21 How do you know they have the same  
22 characteristics?

23 MR. WHITLOCK: That's what your question  
24 said.

1           A.    Yes.

2                   MR. WHITLOCK:   You are asking him to make  
3   assumptions.

4                   MR. WOLFF:    Jamie, if I wanted to hear your  
5   answer, I would swear you in.

6           A.    But that was my answer.

7           Q.    My question to you is, how do you know that five  
8   different individuals are otherwise identical in all  
9   respects?

10                  MR. WHITLOCK:   Because you didn't ask him to  
11   make that assumption.

12                  MR. WOLFF:    Jamie, I am not asking you the  
13   question.

14           A.    Well, let me answer it.   You have only given me  
15   this limited information and it is all the same.   So I  
16   have to assume that you want my answer based on that and  
17   my answer is the same risk.

18           Q.    In your experience, are all individuals identical  
19   from individual to individual?

20           A.    Okay.   We have to make something clear here.   You  
21   are talking about individuals, either one, two, five.   I  
22   am an epidemiologist.   I look risk which is population  
23   based.   I am responding to you on the basis of human  
24   populations.   Not on individuals.

1 Q. So your analysis here would be an epidemiological  
2 analysis because you deal with populations of people as  
3 opposed to a clinical medicine prospective which deals  
4 with individuals, is that fair?

5 A. That is fair.

6 Q. Please assume that Subject A develops ulcerative  
7 colitis. What are the causative or predisposing factors  
8 known for ulcerative colitis?

9 A. Very little is actually known.

10 Q. Of those factors that are known, what are they?

11 A. I mean, age is a factor because it is a disease  
12 that often times develops in young people. But then  
13 there is as far as I remember a second peak later on in  
14 life where the incidents increases again. So it is age  
15 dependent. Whether there is a difference between men  
16 and women, I don't remember. I don't think it is very  
17 big. There may be one.

18 Q. Anything else that you would put on the list?

19 A. Well, I mean, one of the issues that is currently  
20 being researched is the microbiome. There have been  
21 done, studies have been done of changing the microbiome  
22 with fecal transportation. So it looks like this are  
23 aspects of microbiome that plays a role for the  
24 causation of that disease. But what they are is

1 currently unclear.

2 Q. Anything else that you would put on that list?

3 A. I believe that there is a genetic component as  
4 well. That's all I can remember at this point.

5 Q. Of those factors, which is the highest probable  
6 causative or predisposing factors?

7 A. Again, I would have to look that up.

8 Q. What is the probability of PFOA as the causative  
9 factor?

10 A. I believe that there is substantial evidence that  
11 it is.

12 Q. What is the probability numerically?

13 A. We don't know exactly what the probability is.  
14 It is like a judgment call that the evidence is  
15 substantial; but I do agree that we should try to get  
16 more research on this and I am doing research on this.  
17 So I hope that we can contribute some more evidence.

18 Q. Are any other chemical or physical agents  
19 potential causes of ulcerative colitis?

20 MR. WHITLOCK: Objection to the form.

21 A. Again, I would have to look it up.

22 Q. Sitting here today, you do not know, is that  
23 fair?

24 A. Yes. That's fair enough.



1 Q. Please assume that Subject B develops  
2 hypercholesterolemia, what are the predisposing factors  
3 known for hypercholesterolemia?

4 A. First of all, there is a genetic, a mutant that  
5 predisposes to what is called familial  
6 hypercholesterolemia that is easily recognized because  
7 the concentrations are really, really high. So that's a  
8 genetic component. There are obviously dietary and  
9 physical activity aspects of that.

10 Q. Sure. Of those factors, what is the highest  
11 probable causative or predisposing factor for the  
12 hypercholesterolemia in Subject B?

13 MR. WHITLOCK: Objection to the form.

14 A. The point is that genetic can be very strong and  
15 it is not that it is very common, but it is just that  
16 when you see it, the concentrations are so high. So  
17 that's a strong factor.

18 Q. What is the probability of PFOA as the causative  
19 factor?

20 A. Again, I think the evidence is substantial.

21 Q. Can you quantitate that?

22 MR. WHITLOCK: Objection to the form.

23 A. What one can do, but it has not been done yet,  
24 what, a meta analysis of the data that are available,

1 but not all of the study are as well conducted as they  
2 should. So one would have to go into detail to assess  
3 the, you know, variety of studies that are available.  
4 There are several.

5 Q. Do you know how to conduct a meta analysis?

6 A. I know and I have done that in the past, but I  
7 didn't think is that I should spend my time doing that  
8 for this purpose.

9 Q. So just to be clear, you have not performed a  
10 meta analysis with respect to PFOA exposure and any  
11 health endpoint; is that correct?

12 A. That's correct.

13 Q. Are there any other chemical or physical agents  
14 that are potential causes of hypercholesterolemia?

15 A. I believe so. I don't remember any of them.

16 Q. Please assume Subject C develops thyroid disease.  
17 What are the causative are predisposing factors known  
18 for thyroid disease?

19 A. I mean, there are several types of thyroid  
20 disease. Certainly, there are factors such as  
21 radiation. Say it is radioactive iodine, that's  
22 certainly a risk factor that we are very much aware of.  
23 Particularly in the Chernobyl area.

24 Q. What are the other predisposing or causative

1 factors besides radioactive iodine?

2 A. I mean, I teach this. I usually use a  
3 biomechanical depiction of what happens in the thyroid  
4 gland and then I have a variety of chemical compounds.  
5 How they interfere with this iodine, it interferes with  
6 the enzyme and that. Dioxin is one. There is some  
7 pesticides that interfere.

8 Q. What pesticides?

9 A. I would have to go back and refer to that. I did  
10 not prepare for that today.

11 Q. Of those factors, which is the highest probable  
12 causative or predisposing factor?

13 A. You know, that depends on the level of the  
14 exposure. Dioxin is a risk factor. Dioxin exposure has  
15 gone down. So I doubt that dioxin any longer is a major  
16 factor. Likewise with radioactive iodine far from  
17 Chernobyl.

18 Q. What is the likelihood that the thyroid disease  
19 is idiopathic?

20 A. The word idiopathic means that there is no known  
21 cause of it. It is a difficult word that physicians use  
22 when they don't know what to say. So the word  
23 idiopathic means thyroid disease of unknown cause. It  
24 is not very helpful.

1 Q. What is the likelihood that the thyroid disease  
2 has an unknown cause?

3 A. I think that's very substantial.

4 Q. What is the probability of PFOA as the causative  
5 agent?

6 A. Again, I refer to thyroid disease in my report  
7 and I think I used the word substantial evidence.

8 Q. Can you give me a point estimate for the relative  
9 risk?

10 A. No, I can't. Because that type of detail does  
11 not yet exist.

12 Q. Please assume that Subject D develops kidney  
13 cancer. What are the causative or predisposing factors  
14 known for kidney cancer?

15 A. Well, I should say that the most common form of  
16 kidney cancer, and I think that's what you refer to  
17 hyperlymphoma. Then we would have to refer to that and  
18 note metastatic cancer. Anyway, that disease is to a  
19 substantial degree of unknown origin. There is very  
20 little occupational, for example, evidence of particular  
21 chemicals causing hyperlymphoma.

22 Q. What's the probability PFOA is the causative  
23 agent?

24 A. I have the same answer again. That there is

1 substantial evidence with the exact probability in part  
2 unknown and in part also depends on the subject who gets  
3 the cancer. Because other factors may play a role as  
4 one age. That is the only way I can answer that  
5 question.

6 Q. Finally, let's talk about Subject E. Assume that  
7 Subject E develops testicular cancer. What are the  
8 predisposing factors known for testicular cancer?

9 A. Well, there are different forms of testicular  
10 cancer. At least there is one form that occurs at a  
11 younger age and then picks up later on in life.  
12 Although we don't know the details yet, it looks like.  
13 So we do not know the details yet. There is at least  
14 one prominent factor that has been researched in recent  
15 years and that is inter uterine exposure to endocrine  
16 disrupters, particularly androgen disrupters

17 Q. Is that some sort of an endocrine disorder?

18 A. No. It is because when the fetus is like eight  
19 or nine weeks, that is when the sexual differentiation  
20 begins. Otherwise, all humans will become women. So  
21 there has to be the correct imprinting happening and for  
22 the male it is that testicular cells develops so they  
23 can generate testosterone that then tricks that  
24 development. If there is some interference, and we

1 don't know if PFOA does it, but it is possible if that  
2 interference happens from an exogenous cause, then s  
3 that can cause abnormalities which include testicular  
4 cancer.

5 Q. What is the probability of PFOA as the causative  
6 agent?

7 A. I believe it is substantial as I have written in  
8 my report.

9 Q. Can you give me a relative risk point estimate  
10 for that?

11 A. Again, I have to refer to my previous question  
12 that that depends on the circumstances, the age, level  
13 of the exposure; but there is no doubt, well, I will say  
14 there is substantial evidence that testicular cancer is  
15 an effect that can be attributed to PFOA.

16 Q. What is the relative risk range?

17 A. Again, I can't tell you what the exact relative  
18 risk is.

19 Q. I'm not asking exact. What's the range?

20 A. I can't. I would have to sit down and look at  
21 the exposure levels and do some calculations. I can't  
22 do that on the spot.

23 Q. Is there a methodological difference between  
24 identifying risk of an adverse effect from an

1 epidemiological study and identifying the cause of an  
2 adverse event in a clinical trial?

3 A. There is. In the clinical trial as I understand  
4 it, it is a controlled trial with blinding, where you  
5 control the exposure. So we are back to the question  
6 you asked before that we essentially in the clinical  
7 trial conducting an experiment where we know exactly the  
8 dosage, but that's not what we do in exposure studies.  
9 For example, of the Vermont population exposed to PFOA,  
10 where we can only evaluate the exposure after the fact.

11 Q. Are there any other methodological differences  
12 between identifying a risk of an adverse effect from an  
13 epidemiological study and identifying the cause of an  
14 adverse event in a clinical trial?

15 A. I think we already touched upon the major issues.  
16 If there is anything specific, I am happy to comment on  
17 that.

18 Q. I want to make sure we are not leaving anything  
19 on the table. Thanks.

20 The studies that you cite on Pages 27  
21 through 62 of your report are not the totality of the  
22 published studies from PFOA exposed human populations on  
23 each of those endpoints, are they?

24 A. No. They are, of course not, all of the studies,

1     those are the major studies.

2           Q.   What were your inclusion and exclusion criteria  
3   if any for the studies that you cite on Pages 27 to 62  
4   of your report?

5                   MR. WHITLOCK:   Objection.   Asked and  
6   answered.

7           A.   What I did was to look through the complete  
8   evidence essentially what I had access to.   There may  
9   have been some in languages that I don't speak that I  
10   have missed, but I looked at the accessible literature  
11   and I also looked at FSA and ATSDR, and what I did was  
12   to select the literature that I thought contributed to  
13   the evaluation of a possible association of the outcome  
14   with PFOA exposure.

15          Q.   In your opinion, how long would a person have to  
16   drink water containing PFOA to be at an significantly  
17   increased risk of a serious latent disease?

18                   MR. WHITLOCK:   Objection to the form.

19          A.   Right.   So you are not giving me the full  
20   information, because I would ask for the PFOA  
21   concentration in the water.   I think that's very  
22   important to judge whether there is more than a  
23   background risk.   So what we talked about here, PFOA  
24   exposure from the drinking water in the class, I assume



1 this is what we talked about, well, because any way, and  
2 we talked about residents. So they are not just having  
3 exposure during their vacation, but they are residents.  
4 We are talking about longer term exposure and that  
5 totality in my mind adds to a clearly elevated risk,  
6 elevated exposure, I should say. Therefore an elevated  
7 risk.

8 Q. In your opinion, how long would a person have to  
9 drink water containing 70 parts per trillion of PFOA to  
10 be at an significantly increased greater risk of a  
11 serious latent disease?

12 A. I am not prepared to answer that. Because I  
13 would want to make some calculations before I answer.

14 Q. In your opinion, how long would a person have to  
15 drink water containing 140 parts per trillion of PFOA to  
16 be at increased risk?

17 A. The same answer.

18 Q. If I was to ask you five or ten or one thousand  
19 parts per trillion, your answer would still be the same;  
20 is that correct?

21 A. It would because it is all on a relative scale.  
22 I think this is a question that has to be settled by the  
23 court. It is not my medical opinion that is important  
24 here.

1 Q. Sorry. What's the issue that has to be decided  
2 by the court?

3 A. If a subject who has, if a subject has to have an  
4 exposure of a thousand like you mentioned in order to be  
5 approved as a member of a class by the court, that's not  
6 up to me.

7 Q. That's not what I am asking. I am not asking you  
8 for who is included in a class definition or not. I am  
9 asking from a medical and scientific perspective. Not a  
10 legal one.

11 A. Right. Okay. I can then give you a flat answer  
12 which is sort of very general. That is my opinion.  
13 That is does all of the water in that area contain a  
14 toxic and manmade substance that has nothing to do  
15 there. According to my calculations, I need to have  
16 very, very low water levels, below one, as we discussed  
17 before in order to be safe.

18 Q. I appreciate that you discussed that. My  
19 question is a little bit different.

20 How long would a person have to drink water  
21 containing PFOA, whether it is at five parts per  
22 trillion, ten parts per trillion, a hundred parts per  
23 trillion or a thousand parts per trillion to be at a  
24 significantly increased risk of a serious latent

1 disease?

2 MR. WHITLOCK: Objection to form. Calls for  
3 legal conclusion.

4 A. Again, I am a scientist. I have to refer to what  
5 I have studied and what I have read myself. I have done  
6 the calculations saying that the levels should be below  
7 one and now you mentioned to me some very high levels in  
8 comparison in asking me to say how long does it take. I  
9 can't do that calculation offhand.

10 Q. How long would a person have to drink water  
11 containing one part per trillion of PFOA? In your  
12 opinion, how long would a person have to drink water  
13 containing one part per trillion of PFOA to be as a  
14 significantly increased risk of a serious latent  
15 disease?

16 MR. WHITLOCK: Objection to form. Calls for  
17 legal conclusion.

18 A. Again, I have suggested that the level should be  
19 below one. Now, you are giving me a number just before  
20 that and I think that it is a theoretical issue of what  
21 the actual risk may be. It is clear that it will be  
22 less than at seventy but it is not zero.

23 Q. How long would a person have to drink water  
24 containing just less than one part per trillion of PFOA

1 to be at a significantly increased risk of a serious  
2 latent disease?

3 MR. WHITLOCK: Objection.

4 A. I think I answered this in various ways. I have  
5 to refer to my previous answer.

6 Q. If there is a risk associated with drinking water  
7 with PFOA, then that risk is different from individual  
8 to individual because their consumption of water is  
9 different; is that correct?

10 A. The consumption level, well, of course, in fact  
11 the uptake at the same concentration in the water and  
12 then that would then also relate to differences in risk.

13 Q. Assuming there is a risk associated with PFOA  
14 exposure, if you were to look at any individual putative  
15 class member among the thousand or more individuals in  
16 this proposed class, the risk to that individual is  
17 going to be affected not only by the amount of water but  
18 also by the amount of PFOA in the water he or she has  
19 consumed; is that correct?

20 A. Exactly.

21 Q. Is it fair to say that the risk of disease if any  
22 is not proportional to the difference in the amounts of  
23 water consumed because different individuals have  
24 different susceptibilities to PFOA based on their

1 medical conditions or other behavioral factors?

2 A. I would not express it that way. I would say  
3 that of course there are factors such as sex and age  
4 that play a role, but I am looking at this from a  
5 population viewpoint, and the greater the exposure, the  
6 greater the uptake, the greater the blood concentration,  
7 the greater the risk. That is my best answer to that  
8 question.

9 Q. Is it fair to say that individuals who are not  
10 exposed to PFOA through their home's water supply  
11 because for example they are hooked up to the municipal  
12 water system in Bennington or because they have  
13 activated carbon filter systems that they passed their  
14 water through, do not have that component of PFOA in  
15 their body burden and thus their risk of exposure if any  
16 is less than those individuals who have PFOA in their  
17 home water drinking supply?

18 A. And they are drinking the same amount about, and,  
19 yes. Of course, I mean, again, I have to give you my  
20 same answer. The risk is not nil but clearly there are  
21 relative differences.

22 Q. Is an alteration of a biochemical marker itself a  
23 clinical pathology?

24 A. This is a matter of judgment. This is something

1 that regulatory agencies are very much into. As a  
2 scientist, it is a whole lot easier to look at markers  
3 because everybody has an enzyme at some level or  
4 whatever that mark is, but it is not everybody that has  
5 fatty liver disease.

6 Q. In your opinion, are small but statistically  
7 significant increase in liver biochemical tests  
8 clinically significant?

9 MR. WHITLOCK: Objection to the form.

10 A. It depends. If it occurs, you can discuss that  
11 without the reference interval or if it is high, let's  
12 say, a young person or a child, it may be more  
13 significant than if it perhaps is for an elderly person  
14 who has already been diagnosed hypercholesterolemia. It  
15 is really, I have to mention caveats like that.

16 Q. In your opinion, does every instance of an  
17 abnormal liver biomechanical test constitute a disease?

18 A. I can't think of an example that it does, because  
19 in my, according to my knowledge of clinical matters in  
20 a diagnosis is never, oh, I can't say that because that  
21 is not true. A clinical diagnosis would rely on some  
22 type of clinical findings that are usually beyond just  
23 clinical pathology block tests.

24 Q. So would you agree even if an individual's liver

1 panel test results are different from the normal value,  
2 that the individual may not have a problem?

3 A. Again, that's hard to say, because it could  
4 perhaps be some genetic or constitution that makes this  
5 person to be high in particular liver enzymes. I mean,  
6 I would not as a physician rely on that solely.

7 Q. What else would you rely on?

8 A. I would rely for example on a liver biopsy, on a  
9 scanning. I would rely on a whole battery of clinical  
10 tests. You can also, you can assess crudely the size of  
11 the liver.

12 Q. Is there any risk of performing a liver biopsy?

13 A. There is of course a risk associated with that.  
14 So this is what you like to do when there is a  
15 substantial likelihood that there is something to find.  
16 This is not something that I would ever do in a  
17 population study of a healthy subject.

18 Q. On Page 28 of your report in the fifth paragraph.  
19 You write, my review of available epidemiological  
20 studies demonstrates a strong link between PFAS exposure  
21 and adverse effects on human immune system functions.  
22 Do you see that?

23 A. Yes.

24 Q. What is a strong link?

1       A. Well, it is another way of saying that there is  
2 substantial evidence.

3       Q. Is the phrase strong link synonymous with a cause  
4 and effect relationship?

5       A. It is pretty close. It certainly is stronger  
6 than a possible link that we talked about before.

7       Q. It is close but not quite?

8       A. This is a matter that would have to be discussed  
9 with, you know, whoever the recipient is. If this is  
10 the US EPA, for example, if they would regard the  
11 evidence strong enough to call it causal, and I would  
12 not preclude what EPA would decide on that.

13       Q. As you use the phrase strong link, are you using it  
14 to denote a causal relationship?

15       A. I didn't use that word. I am saying that there  
16 is substantial evidence. That is as close as I can get  
17 at this point.

18       Q. Doctor Ducatman is not proposing monitoring of  
19 serum antibodies or any immuno globulin concentrations,  
20 is he?

21       A. I think you are correct. I don't remember it by  
22 heart.

23       Q. Doctor Ducatman is not proposing monitoring for  
24 immune toxicity or immuno suppression, is he?



1       A. Again, my answer is the same. I think you are  
2 correct.

3       Q. On Page 34 in Paragraph E of your report, you  
4 write I conclude that the human evidence strongly  
5 supports the existence of PFAS dependent immuno toxicity  
6 background exposure levels. Correct?

7       A. That's what I said here.

8       Q. Is it your opinion that the average American  
9 is experiencing immuno toxicity as you term it?

10      A. I would say so.

11      Q. Is an alteration of an immunological biomarker  
12 itself a clinical pathology?

13      A. It depends on what you consider clinical  
14 pathology. I consider it a marker of immune dysfunction  
15 where we don't quite yet understand the reach of that  
16 dysfunction to which degree this, for example, might be  
17 linked to greater cancer risk. Because the immune  
18 system is involved in removing abnormal cells. We don't  
19 know that yet.

20      Q. We don't know?

21      A. This is what IARC says, is this the possible  
22 mechanism. We don't know the mechanism for PFOA to what  
23 causes cancer. But it is possible that the immune  
24 system could play a role.

1 Q. Is it fair to say for example that a reduction in  
2 an individual's diphtheria antibody concentration does  
3 not mean that individual will necessarily contract the  
4 diphtheria?

5 A. That's correct. But you have also to consider  
6 that we did not look at that antibody as such. We  
7 looked at the T cell dependent B cell function and  
8 plasma cell generation of the specific antibodies. So  
9 as an integrated function of the immune system, what we  
10 found in the immune system was deficient. So we were  
11 not interested in diphtheria.

12 Q. Let me ask you a couple of questions. I think we  
13 may have covered this before. If we have, I apologize.

14 A. No problem.

15 Q. In the last paragraph on Page 10 of your report,  
16 when you make reference to Bennington residents who have  
17 an accumulation of PFOA in the body at above background  
18 levels, you are referring there to residents who exceed  
19 2.1 micrograms per liter, correct?

20 A. That's what I say here.

21 Q. How can one tell which Bennington residents do or  
22 do not have more than 2.1 micrograms per liter of PFOA  
23 in their blood?

24 A. I can only guess, but I took this information

1 from the case definition or rather, what is it called,  
2 the class definition.

3 Q. So my question is a little bit different than  
4 that.

5 Looking at the Bennington population at  
6 issue, which are some one thousand or so individuals,  
7 how can somebody tell whether any particular residents  
8 do or do not have more than 2.1 micrograms per liter of  
9 PFOA in their blood, how would one know that?

10 A. You are asking an epidemiologist, and I would say  
11 it is a great majority of those people who would have  
12 more than 2.1.

13 Q. How would one determine that?

14 A. I would assume that, I mean, this is not, I have  
15 not been into this. The way to figure that out is to  
16 take a blood sample and analyze it.

17 Q. Fair enough. We touched upon this very briefly  
18 before. You are aware that NHANES periodically conducts  
19 and publishes results of blood sampling; is that  
20 correct?

21 A. I understand that very well.

22 Q. Is it fair to say NHANES collects and publishes  
23 data on human exposure to some 265 environmental  
24 chemicals?

1       A. I don't remember the exact number, but that  
2 sounds right.

3               MR. WOLFF: Mark this as Exhibit Number 11.  
4               (PFOA blood data tables was marked as  
5 Exhibit Number 11 for identification)

6       Q. Exhibit 11 contains the PFOA blood data tables  
7 from the February 2015 NHANES fourth national report on  
8 human exposure to environmental chemicals. If you  
9 would, please, turn with me to Page 338 which contains  
10 the sampling results for PFOA from 2011 to 2012. Are  
11 you there?

12       A. Yes.

13       Q. Top line, the geometric mean for the total sample  
14 is 2.08, which rounded up to the first decimal is the  
15 number that you have been using and we have been using,  
16 which is 2.1 micrograms per liter; is that correct?

17       A. That's correct.

18       Q. If you would please turn to Page 336, which  
19 contains the sampling results for PFOA from five  
20 different surveys taken between 1999 and 2010; is that  
21 correct?

22       A. Yes.

23       Q. With the exception of the 2007 to 2008 sampling,  
24 in each successive survey, the geometric mean is going

1 down from a prior survey; is that correct?

2 A. That's correct.

3 Q. For 1999 to 2000, the geometric mean was 5.21  
4 micrograms per liter; is that correct?

5 A. Yes.

6 Q. As we know, for 2011, 2012, the geometric mean  
7 was 2.08; is that correct?

8 A. Yes.

9 Q. Since you say that anybody in this class who has  
10 PFOA blood serum levels above 2.1 would require medical  
11 monitoring, would you say that millions and millions of  
12 Americans should have been getting this medical  
13 monitoring as a medical necessity in 1999 to 2000 when  
14 background level was 5.21 which is two-and-a-half times  
15 higher than 2.1?

16 MR. WHITLOCK: Objection to the form.

17 A. I have to correct you because I am not saying  
18 that. You should refer to that paragraph that you  
19 referred to before on Page 10. The bottom paragraph.  
20 Because I am referring to the class information. That's  
21 not mine. What I am saying is that this population is  
22 and has been exposed to an elevated exposure and  
23 therefore elevated risk and because it is under those  
24 local circumstances, I am saying that elevated risk

1     should trigger medical monitoring. So I am not talking  
2     about all the United States here.

3           Q. Let me ask you this. Would you say that millions  
4     and millions of Americans should have been getting this  
5     medical monitoring as a medical necessity in 1999 to  
6     2000 when the background level was 5.21 micrograms per  
7     liter?

8           A. I have never considered that question, but I can  
9     tell you that one of the reasons is that these amounts,  
10    these concentrations and serum is so high is that we  
11    know water has been contaminated across the United  
12    States on numerous sites and we know that at least in  
13    one of the studies that we did, five or six million  
14    Americans are exposed to elevated concentration of  
15    PFASes in drinking water. So parallel to the Vermont  
16    case and those numbers are included here. That's why  
17    this is not really a proper background. It is actually  
18    too high. Maybe it was even much too high in the past.

19          Q. I guess my question is, if anybody in a  
20    population has got 5.2 micrograms per liter of PFOA in  
21    their blood, would you say that they need medical  
22    monitoring as a medical necessity?

23          A. I would not say that because that depends on the  
24    circumstances. I understand the circumstances here are

1 quite specific in that there is a local source, and we  
2 know exactly comes through drinking water. We know  
3 these are resident who have been exposed to this for  
4 quite a while.

5 Q. If the exposure level is the same from an  
6 individual in Bennington and an individual in Peoria,  
7 Illinois, they have the same identical blood levels,  
8 what difference does that make as to whether they  
9 require medical monitoring for potential future disease?

10 A. I can't answer that. Medical monitoring in this  
11 particular case has been defined as it is here and all I  
12 am saying that is reasonable. I can support that. And  
13 the way that Doctor Ducatman has described it, I am in  
14 favor of that. I have not been asked to look the state  
15 of Illinois and specific locations. I would give you  
16 that those elevations and serum concentrations will  
17 result necessarily in an increased risk. That relates  
18 to my expert report here.

19 Q. I guess what I am sort of having trouble getting  
20 my head around is if a person has got 5.1 micrograms per  
21 liter of PFOA in his or her blood in California or  
22 Arizona or Texas or Missouri or Mississippi or New  
23 Jersey or Vermont or New Hampshire, why should the  
24 particular location where that person resides make a

1 difference from a clinical perspective as to whether  
2 they need medical monitoring for exposure to PFOA?

3 A. I completely understand your question. All I can  
4 say, I didn't comment on medical monitoring. I did look  
5 at Doctor Ducatman's report and I think he argued that  
6 convincingly. I have not seen similar considerations on  
7 other locations in the United States. I do insist that  
8 they have an increased risk as well, but whether that  
9 should trigger perhaps a completely different  
10 circumstances of medical monitoring, I have no opinion.

11 Q. Have you ever expressed an opinion that any  
12 particular population requires a medical monitoring  
13 program?

14 A. I don't remember so in this regard, I don't think  
15 so.

16 MR. WOLFF: Mark is this last Exhibit Number  
17 12.

18 (Transcription was marked as Exhibit Number  
19 12 for identification)

20 Q. So Exhibit 12 is a transcription of the ATSDR,  
21 CDCs January 2017 PFAS continuing education for  
22 clinicians, which is available on video through the  
23 ATSDR's website, and which contains subtitles. I just  
24 want you to be clear about this document. This document



1 was prepared by our word processing department using the  
2 transcription off of that video and its subtitles.

3 A. Okay.

4 Q. Please, turn with me to Page 28. As recently as  
5 2017, the ATSDR, CDC has told clinicians that there is  
6 no health screening recommended because of exposure to  
7 PFOA; is that correct?

8 A. I see that.

9 Q. Please, turn with me to Page 27. We are going to  
10 take this in stepwise fashion. As recently as 2017, the  
11 ATSDR and CDC has told clinicians that PFAS are  
12 ubiquitous in both the United States and globally.  
13 There are no specific biomarkers of health affects  
14 caused by or linked to PFAS blood concentrations. The  
15 presence of PFAS in blood testing only confirms exposure  
16 which is present and greater than 95 percent of the  
17 United States population based on representative samples  
18 from the NHANES studies; is that correct?

19 A. I see that.

20 Q. Do you agree with that statement?

21 A. I agree that PFAS is ubiquitous in the United  
22 States. It says there are no specific biomarkers of  
23 health effects. I wonder what they referred to. It  
24 just says biomarkers. I would like to know what

1     biomarkers they refer to. And the presence of PFAS in  
2     blood confirms exposure. Well, that is a standard  
3     expression from government or authorities. While higher  
4     blood --

5           Q. I am getting there next. I did not get there  
6     yet.

7                   MR. WHITLOCK: What page are you on?

8                   MR. WOLFF: 27.

9           Q. Let's take that next statement, Doctor. The  
10    ATSDC, CDC further state, while higher blood  
11    concentrations of PFAS express larger exposure, PFAS  
12    blood concentrations cannot be linked to any specific  
13    health effects and results obtained from testing  
14    patients, blood PFAS concentrations would not guide  
15    medical decisionmaking; is that correct?

16          A. I see that, but you have to recognize that that  
17    is a statement on behalf of the Centers for Disease  
18    Control, which is always trying to, you know, provide a  
19    statement that cannot be upsetting to anybody. This is  
20    a type of statement that they will say about fluoride in  
21    drinking water. This is just their interpretation which  
22    has, you know, different, defensive, so to speak aspects  
23    in addition to the science. It is not just a pure  
24    summary of the science.

1 Q. Let's go to the next statement. The ATSDR CDC  
2 further states that even if a patient is identified as  
3 having an extremely high PFAS blood concentration, this  
4 does not mean he or she will suffer from any adverse  
5 health affects; is that correct?

6 A. That's what it says. It is sort of a strange  
7 statement.

8 Q. Do you agree with that statement?

9 A. I can agree in the sense that we talked about  
10 before that on average, the risk is higher. But the  
11 fact that there is an elevated risk does not mean that a  
12 specific individual will develop specifically that  
13 disease.

14 Q. The ATSDR, CDC further states that, likewise,  
15 patients with mildly elevated PFAS blood concentrations  
16 are not immune from exposure related health risks.  
17 Management of patients exposed to PFAS should be guided  
18 solely by patient symptoms and findings derived from a  
19 thorough health history and physical examination; is  
20 that correct?

21 A. It is correct that that's what it says.

22 Q. My next question is, do you agree with that  
23 statement?

24 A. I agree with it to the extent that I would take

1 into account the PFAS exposure.

2 Q. Do you agree with or disagree with anything else  
3 in that statement?

4 A. This very last, the management?

5 Q. The very last one.

6 A. I have difficulty with the one where they say the  
7 management should be guided solely by the patient's  
8 symptoms and findings deriving from a thorough physical  
9 health history and physical examination. That is not  
10 enough. For example, that could be physician reports on  
11 something else and that's not just a health history.  
12 That could be occupational exposure. Similar things. I  
13 would not, what you said, this has been taken off a  
14 tape. I think this is not a proper complete statement  
15 and I don't think CDC would give you that impression  
16 that it is.

17 Q. Please turn with me to Page 32. Are you there?

18 A. I am there.

19 Q. The atsdrcdc states that more than 95 percent of  
20 the represented United States population has measurable  
21 blood levels of PFOA and PFOS. The presence of these  
22 PFAS in blood only confirms exposure. This does not  
23 mean your patient will suffer any adverse health  
24 effects. Routine blood test for PFAS cannot be

1 extrapolated to any specific health effects and cannot  
2 guide medical decisionmaking; is that correct?

3 A. That's what it says.

4 Q. Do you agree with that statement?

5 A. No. In the present situation, Number 1, we know  
6 more than they knew when they wrote this up, and what  
7 they decided to say on that occasion later on in January  
8 two years ago. So that's one aspect. I think that, I  
9 mean, the statement that your patient will not  
10 necessarily, they are missing the word necessarily,  
11 suffer from any adverse affects. That has to be  
12 interpreted properly in that people who have elevated  
13 exposures to PFAS have necessarily an elevated risk, and  
14 in the situation in Vermont where you have the zone of  
15 contamination, you have a confined population. This is  
16 where we have been arguing about medical monitoring.

17 Q. Is it fair to say that because of biomonitoring  
18 sampling results for PFOA cannot predict current or  
19 future health outcomes or diseases, the results are not  
20 clinically actionable?

21 A. Again, I would say, we should ask CDC if that is  
22 our view today, because it does not sound right that CDC  
23 would issue that as guidance to physicians of this  
24 country. They should know better today. Also, given

1 the most recent ATSDR report.

2 MR. WOLFF: Let's take a break. Off the  
3 record.

4 THE VIDEOGRAPHER: The time is 2:45 p.m. We  
5 are going off the record.

6 (Whereupon a break was taken)

7 THE VIDEOGRAPHER: We are back on the  
8 record. The time is 2:56.

9 Q. So during the break, counsel had a discussion  
10 sort of flush out the parameters of the stipulation from  
11 the plaintiffs' counsel. So while we understand that it  
12 has been stipulated that the plaintiffs are not  
13 proffering you as an expert in medical monitoring, and  
14 as a result of that, we are going to forego questioning  
15 about individual blood tests, specificity, sensitivity,  
16 positive predictive value in the population, that you  
17 are nevertheless endorsing as a general principle Doctor  
18 Ducatman's recommendation for a medical monitoring  
19 program, at least conceptually; is that correct?

20 A. It is reasonable and pragmatic and I think  
21 justified.

22 Q. Is it fair to say that medical monitoring in the  
23 form of medical screening is a systematic search for a  
24 disease in people who have no signs or symptoms of that

1 disease?

2 A. I think that is reasonable.

3 Q. When you commit asymptomatic patients to medical  
4 monitoring, you are making a medical decision and are  
5 intervening in their lives medically; true?

6 A. It depends on what you think to intervene. It is  
7 a medical intervention. But the degree to which it  
8 intervenes with peoples' lives is a matter of judgment.

9 Q. In an asymptomatic population, is it fair to say  
10 that the vast majority of those people being screened  
11 will not have the disease being sought?

12 MR. WHITLOCK: Objection to the form.

13 A. That may be true. It may be different here in  
14 Bennington. Because people have an elevated exposure to  
15 PFOA.

16 Q. Is it fair to say that medical screening in an  
17 asymptomatic population is often up against staggering  
18 odds?

19 MR. WHITLOCK: Objection to the form.

20 A. I don't know what staggering odds are. If this  
21 can be conducted with the purpose of finding cases early  
22 so that they can benefit from medical intervention at a  
23 stage where there is a substantial benefit to gain, I  
24 would say this is a good idea.

1 Q. I am not fussing when I say that, but there are a  
2 lot of ifs that are baked to that statement, true?

3 A. The way you state it, that is correct.

4 Q. Is it fair to say that if an asymptomatic  
5 population is being screened, it must involve many  
6 people to potentially benefit few?

7 MR. WHITLOCK: Objection to the form.

8 A. Again, that depends on the situation. It may be  
9 true on some situations but not on others.

10 Q. In assessing the utility of the medical  
11 monitoring program in an asymptomatic population,  
12 wouldn't one have to take its risks and benefits into  
13 account?

14 A. That would be normal.

15 Q. Is it fair to say that in a population of a  
16 thousand asymptomatic individuals being screened over a  
17 decade in an older population and even with a common  
18 cancer, only a few may be ten to twelve would be  
19 destined to die from a cancer?

20 A. That sounds reasonable but as a physician, I have  
21 to insist that every disease of subject, say, in among  
22 the residents in the zone of contamination is one we  
23 didn't do enough for to save.

24 Q. Is it fair to say that in assessing a medical



1 monitoring program in an asymptomatic population, the  
2 population being screened should be at a significantly  
3 high risk for the undiagnosed disease, that is, the  
4 disease should have a sufficiently high prevalence in  
5 the population?

6 A. Again, you are using the words significant and  
7 sufficient that are sort of, I don't understand what  
8 they mean, because you are speaking in general terms,  
9 but I understand what you are aiming at and I agree with  
10 that. Not necessarily with the specific way that you  
11 said it.

12 Q. Is it fair to say that in assessing medical  
13 monitoring program in an asymptomatic population, that  
14 the disease being screened for should have a  
15 sufficiently high prevalence in that population?

16 A. When you say sufficient, it is again a matter of  
17 consideration, and, you also, you continue to say the  
18 whole population is asymptomatic. That is very  
19 unlikely. So what we are dealing with here, we are  
20 dealing with an exposed population which must have an  
21 elevated risk. So this is why it is reasonable to  
22 consider medical monitoring to catch those cases that  
23 can be caught early so they can benefit from medical  
24 intervention.

1 Q. Yet, sitting here today, you cannot quantify what  
2 that elevated risk might be for any particular endpoint  
3 in terms of giving a relative risk point estimate or 95  
4 percent confidence intervals around the point estimates,  
5 can you?

6 A. We are talking two things here. I am capable of  
7 doing that, but I can't do it right here on the spot and  
8 especially with the little information you give me.

9 Q. Well, I put no constraints on the scope of your  
10 report. Your report does not provide that information?

11 A. What information? What the relative risk is?

12 Q. Yes.

13 A. No, it does not because that is something that  
14 would depend on the circumstances like on the degree of  
15 exposure, duration, etcetera. That was not what I was  
16 asked to do. It is nothing that I would be able to do  
17 in a jiffy.

18 Q. Is it fair to say that in assessing a medical  
19 monitoring program in an asymptomatic population, one  
20 should consider the natural history of the disease and  
21 the evidence for an improved clinical outcome as a  
22 result of the medical monitoring for the given disease?

23 MR. WHITLOCK: Objection to the form.

24 A. What you refer to is the material that I have

1     been teaching myself. I can only agree. I would still  
2     comment on your insistence that it is a nonsymptomatic  
3     population. I don't think that is likely.

4           Q. The goal of the detecting disease early through  
5     screening is to improve the outcome when compared to the  
6     outcome of the same disease that is identified because  
7     the patient experiences symptoms and goes to see the  
8     doctor; is that correct?

9           A. Let me hear that again. There were several  
10    components.

11          Q. The goal of detecting disease earlier through  
12    screening is to improve the outcome when compared to the  
13    outcome of the same disease that is identified, because  
14    the patient experiences symptoms and then goes to see  
15    the doctor?

16          A. Compared to those that were identified through  
17    the screening, right, correct.

18          Q. So that's a correct statement?

19          A. It is.

20          Q. Early detection through screening should be known  
21    to have an impact on the natural history of the disease  
22    process, true?

23          A. Again, it depends on what you mean when you say  
24    natural history. I would say the course of the disease,

1 because you aim at identifying at early stage and then  
2 you want to impact that rather than waiting until the  
3 full blown clinical picture.

4 Q. The period of time between when a cancer starts  
5 growing and when it causes symptoms is when screening  
6 can catch the cancer; is that true?

7 A. That is at least the aim of screening.

8 Q. Is it fair to say that one reason there is a  
9 limited benefit to cancer screening is because there can  
10 be cancers that are missed by screening?

11 MR. WHITLOCK: Objection to the form.

12 A. It is part of the story that you can only miss  
13 some. That you can also detect some that were never  
14 meant to become a lethal cancer. So there are issues  
15 like that. It is a little more complicated than you  
16 stated.

17 Q. Aggressive, fast growing cancers are often missed  
18 by screening, aren't they?

19 A. That is one possibility. Yes.

20 Q. What population based cancer screening tests, if  
21 any, have ever been shown to reduce overall mortality?

22 A. I don't remember that by heart. I would refer to  
23 the National Cancer Society or CDC for that information.

24 Q. Are there any?

1           A.    I'm sure.

2           Q.    As a physician, do you subscribe to the  
3    proposition that health is not only a physical state of  
4    being, but is also a state of mind?

5           A.    Well, the WHO definition does not say state of  
6    mind but it refers to mental health as well.

7           Q.    Do you believe that mental health is part of a  
8    person's health status?

9           A.    I believe so.

10          Q.    Do you agree that as a physician, you must be  
11    careful not to undermine a person's state of health?

12          A.    I don't think that is something every physician  
13    would consider doing. It certainly plays a role.

14          Q.    Do you agree that in order for medical screening  
15    tests to be useful, their benefits must outweigh their  
16    risks?

17          A.    I need to understand the word risk here. Are you  
18    referring to the risk, let's say, of a liver biopsy if  
19    that is what is considered necessary for the screening,  
20    is that the kind of risk?

21          Q.    That could be a risk. There could be a risk of  
22    adverse psychosocial sequelae?

23          A.    Fair enough. I agree that one has to take the  
24    whole range of possible risks into consideration as the

1 cost to the individual patients in addition to the  
2 advantages.

3 Q. Do you agree that to determine a test's  
4 usefulness, one should consider its specificity and  
5 sensitivity?

6 A. Yes.

7 Q. Do you agree that in the context of this case for  
8 a test to be useful, early detection must improve the  
9 outcome or the natural history of the disease?

10 A. The question is when you say must improve, we may  
11 not have that evidence available for a population like  
12 this. So it must at least judged to be beneficial.

13 Q. Do you agree that for a test to be useful, it  
14 must have a positive predictive value in a population at  
15 issue?

16 A. Yes. I would say so.

17 Q. Do you agree that knowledge of disease prevalence  
18 is a necessary component of positive predictive value?

19 A. Yes. But the problem is we may not know that  
20 exactly for a population. This may have to be decided  
21 on whatever information that there is available that can  
22 be applied.

23 Q. Is it fair to say that in assessing a medical  
24 monitoring program, one should consider the sensitivity,

1 specificity, and positive predictive value of proposed  
2 medical monitoring tests?

3 A. Yes. To the extent that's possible.

4 Q. Is it fair to say there may be serious  
5 consequences in the use of screening test with poor  
6 sensitivity or poor specificity?

7 MR. WHITLOCK: Counsel, for the record, we  
8 are getting beyond what we agreed upon here.

9 MR. WOLFF: I have not asked about specific  
10 tests like BUN or globulin or albumin, or alkalene,  
11 whatever it was. So I think we are not departing.

12 A. Could you repeat the question? I'm happy to  
13 answer.

14 Q. Sure.

15 Is it fair to say there may be serious  
16 consequences in the use of screening tests with poor  
17 sensitivity or poor specificity?

18 A. The question is in a word serious, what you mean  
19 by serious. Certainly there can be adverse questions.  
20 The circumstances will then define whether they can be  
21 considered serious.

22 Q. What difference is there if for the data required  
23 in determining the presence -- strike that.

24 What difference is there if any for the data

1 required in determining the prevalence of a health  
2 disturbance in a population as opposed to conducting a  
3 risk assessment for a health disturbance in a  
4 population?

5 A. I am a little confused about that. You are  
6 talking about risk assessment. Is that now a risk  
7 assessment for the PFOA or risk assessment for the  
8 procedure? I don't understand.

9 Q. Okay. In terms of an environmental exposure.

10 A. Okay. Like PFOA?

11 Q. Like PFOA.

12 A. Can you repeat the sentence?

13 Q. What difference is there if any for the data  
14 required in determining the presence of health  
15 disturbance in a population as opposed to conducting a  
16 risk assessment for a health disturbance in a  
17 population?

18 A. The first one descriptive epidemiology, you want  
19 to know how frequent it is; and the second one is  
20 analytic epidemiology where you are trying to relate  
21 that to particular suspecting.

22 Q. When you say how frequent it is in the  
23 population, are you referring there to its prevalence?

24 A. That would be the normal, that is, the occurrence



1 at a particular point. That would be the usual measure.

2 Q. In the context of a clinical or a laboratory  
3 test, what is false positive?

4 A. A false positive is one that indicates the  
5 presence of an abnormality where other evidence confirms  
6 or suggests that it is not present.

7 Q. A false positive is a false alarm, isn't it?

8 A. Yes. You could call it that.

9 Q. In the context of a clinical or a laboratory  
10 test, what is a false negative?

11 A. A false negative is one that is missed by the  
12 test despite the fact that other evidence, perhaps even  
13 later on documents that in fact is is present.

14 Q. What is overdiagnosis?

15 A. Overdiagnosis means that there are apparently  
16 included cases of false positives or that subject are  
17 being diagnosed without due justification.

18 Q. Is it fair to say that overdiagnosis is sometimes  
19 referred to as the detection of a cancer that is not  
20 destined to ever cause symptoms or death?

21 A. Right. I have seen literature on that for  
22 example with regard to breast cancer screening. It is  
23 an issue.

24 Q. Is it fair to say that overdiagnosis is the

1 diagnosis of a neoplasm that never would have clinical  
2 significance?

3 A. Well, it depends on the definition of whether you  
4 are, will, accept that a neoplasm has some consequence  
5 but not causing death. It depends on your definition of  
6 the situation. I agree with you in general terms.

7 Q. Is it fair to say that overdiagnosis is the  
8 identification of slow growing cancer that even if  
9 untreated would never cause symptoms or reduce survival  
10 because the screening test cannot distinguish between  
11 the abnormal appearing cells that would become cancerous  
12 from those that would never do so?

13 MR. WHITLOCK: Objection to the form.

14 A. You have just mentioned an example of that. That  
15 could be with regard to breast cancer. I agree that  
16 this is an actual problem that needs to be considered.

17 Q. A false positive test result effectively means  
18 that you are sending a healthy patient to a doctor for  
19 some type of follow-up; is that correct?

20 A. So type of?

21 Q. Follow-up.

22 A. Follow-up, because of a positive test. I assume  
23 that is the case.

24 Q. And a false negative test result, effectively

1 means that you are clearing a patient who should be  
2 referred to a doctor for some type of follow-up?

3 MR. WHITLOCK: Objection to form.

4 A. Right. That would be the proper explanation of  
5 that.

6 Q. What are the harms related to a false positive  
7 test result?

8 A. It depends on the circumstances.

9 Q. What are the harms related to a false positive  
10 test result for kidney cancer?

11 A. It could be a follow-up at the family doctor. If  
12 he also has a false positive, then there could be  
13 additional, therapeutic attempts on that kidney cancer,  
14 but I would assume that rather than the family physician  
15 referring the patient to radiation treatment or  
16 something dramatic, I would assume that there would be  
17 some additional steps so that the false alarm would be  
18 caught in time before the kidney was removed.

19 Q. Is it fair to say that another type of harm from  
20 a false positive is that the patient will become labeled  
21 with having a disease that is not present?

22 A. I don't quite know what you mean by saying label.  
23 Certainly, first of all, the patient can be scared  
24 himself or herself, and that could theoretically be some

1 sort of stigma. Talking about theory here.

2 Q. What are the harms related to a false negative  
3 test result?

4 A. I think we are going over the same ground again.  
5 Clearly, if worse comes to worse, the patient will  
6 unreasonably believe to be free of disease and may not  
7 take proper precautions potentially. So we are all  
8 trying to avoid false negatives clearly because we do  
9 want to do proper diagnosis at the proper time.

10 Q. That's because persons with false negative  
11 results may have delays in diagnosis and treatment; is  
12 that correct?

13 A. That could be the consequence.

14 Q. On the other hand, false positive results can  
15 result in followup testing that is uncomfortable,  
16 expensive, and potentially harmful?

17 A. Potentially. Yes.

18 Q. Is it fair to say that although screening may  
19 prevent the development of disease related morbidity and  
20 mortality, positive test results, both false positive  
21 and true positive may lead to interventions that could  
22 be unnecessary or even risky because of overdiagnosis  
23 and overtreatment?

24 A. That's a theoretical possibility.

1       Q. Is it fair to say the normal ranges for  
2 biomechanical tests are often based on the 95 percent  
3 confidence intervals in a normal healthy population,  
4 that is, although everyone is healthy by convention,  
5 values outside of the 2.5 percent lower and upper  
6 extremes are considered to be abnormal?

7       A. That is the standard practice in biochemistry.

8       Q. It is fair to say that ordering six blood tests  
9 in a normal, healthy individual yields only a 74 percent  
10 chance that all six tests will be normal? In other  
11 words, there is a 26 percent chance that one or more may  
12 be abnormal; is that true?

13       A. On the condition, and you are not expressing the  
14 circumstances properly. Excuse me. If the tests are  
15 independent, you are correct; but oftentimes when for  
16 example the doctor wants to diagnose a liver disease,  
17 the doctor will select six related liver tests and under  
18 those circumstances the calculation is not correct. It  
19 is an exaggeration.

20       Q. With respect to independent tests, when ordering  
21 twelve tests in a normal person, there is a 54 percent  
22 chance that all twelve will be normal and a 46 percent  
23 chance that one or more will be abnormal. Is that true?

24       A. Theoretically, that's true but I don't know of

1 any physicians who will prescribe twelve independent  
2 tests. Because you are always focused on something that  
3 is relevant to the patient's circumstances and it could  
4 be, you know, it PFOA exposure or it could be let's say  
5 yellow sclera that would indicate a liver problem.

6 Q. Do you know how many independent tests Doctor  
7 Ducatman is recommending as part of his medical  
8 monitoring program?

9 A. I have not counted them but I think from my  
10 reading he has selected an appropriate selection of  
11 tests.

12 Q. Do you know whether it is more than twelve or  
13 less than twelve?

14 A. I don't remember.

15 Q. Is it fair to say that simply ordering tests in  
16 healthy individuals or in the absence of clinical  
17 suspicion of a disease may result in many false positive  
18 tests that can lead to false alarms, anxiety, additional  
19 testing, and possible morbidity or mortality from  
20 subsequent testing or interventions?

21 MR. WHITLOCK: Objection to the form.

22 A. What you are saying is theoretically possible,  
23 but I doubt that it is likely in this case.

24 Q. Do you agree that some false positive tests can

1 be in excess of fifty percent such as CAT scan for  
2 sub-clinical malignancies?

3 A. I don't have any specific knowledge so I can't  
4 give you a proper answer.

5 Q. Would you agree that across the spectrum of  
6 screening tests for sub-clinical disorders, it is not  
7 uncommon for the false positive rate to be at least five  
8 percent?

9 A. I would think this is correct; but I don't have  
10 any detailed knowledge myself.

11 Q. Would you agree that for a screening test to be  
12 more likely than not predictive, the positive predictive  
13 value should be greater than 0.5 or in other words  
14 greater than fifty percent?

15 A. I can't comment on that. I don't have any  
16 specific knowledge to relate this to.

17 Q. If we assume that the false positive rate of a  
18 screening test for a sub-clinical disorder is at least  
19 five percent, in order for the positive predictive value  
20 to be greater than 0.5, the prevalence of the disorder  
21 in the population would have to be fifty out of a  
22 thousand, do you agree?

23 A. I have not done the math in my head. The numbers  
24 seem to be okay.

1 Q. Among all the potential disorders you opine on in  
2 your report, is there any single one of them where you  
3 would expect the PFA mediated incidents to be greater  
4 than 0.5 or in other words greater than fifty percent?

5 A. I mean, fifty percent in a prevalence?

6 Q. Yes.

7 A. Prevalence of at least fifty percent, I don't  
8 expect any of them will.

9 Q. You are no doubt familiar with the US  
10 Preventive Services Task Force; is that correct?

11 A. Yes. I am familiar with it; but I can't remember  
12 any quotes that I can relate to.

13 Q. Are you aware that the US Preventive Services  
14 Task Force was established by a congressional mandate  
15 and is comprised of an independent volunteer panel of  
16 sixteen national experts in prevention and evidence  
17 based medicine?

18 A. I don't remember. It sounds correct.

19 Q. Is it fair to say that the task force is  
20 generally considered to be a reputable and reliable part  
21 of the medical community?

22 A. I would assume so.

23 Q. Have you ever been a member of that task force?

24 A. No.



1 MR. WOLFF: Mark this as Exhibit Number 13.

2 (General types of harm for consideration was  
3 marked as Exhibit Number 13 for identification)

4 Q. Exhibit 13 is an excerpt from the US Preventive  
5 Services Task force Procedure Manual. Please turn with  
6 me to Page 43. The second paragraph in Section 6.6.2  
7 under the heading of General Types of Harm for  
8 Consideration. Do you see that?

9 A. I see that.

10 Q. Do you agree with the proposition that harms of  
11 screening may include psychological harm from the  
12 labeling, the harms of diagnostic studies to confirm the  
13 presence of the condition, and overdiagnosis of screen  
14 protected conditions?

15 A. This is a fair statement. I can't disagree with  
16 that.

17 Q. Do you agree with the proposition that because  
18 screening and other preventive interventions are  
19 implemented in asymptomatic persons with the goal of  
20 preventing future disease, one should place a high  
21 priority on considering the harms of overdiagnosis and  
22 overtreatment whereby the preventive service has the  
23 unintended consequence of creating quote, diseases,  
24 close quote, that often leads to unnecessary and

1 ineffective treatment?

2 A. Right. You read this. It is a fair statement.

3 It is not in my report though.

4 Q. Do you agree with the proposition that harms of  
5 early treatment and overdiagnosis may accrued a patient  
6 whose condition may never have come to clinical  
7 attention or for whom the arms of treatment initiated  
8 prior to routine clinical protection were different or  
9 occurred earlier and/or over longer period of time? In  
10 other words, these are harms of treatment that would not  
11 have occurred in the absence of screening.

12 A. These are some general statements that I can't  
13 disagree with.

14 Q. Please turn with me to Page 42. Section 6.6.1.  
15 Are you there?

16 A. I am there.

17 Q. Do you agree with the conceptual notion that  
18 screening is intended for asymptomatic individuals in  
19 order to prevent or delay future health problems?

20 A. That is reasonably stated. Yes.

21 Q. Do you agree with the proposition that the burden  
22 of proof, that the benefits exceed the harms prior to  
23 recommending implementation of screening or other  
24 preventive services is thus higher than it is for

1 diagnosis or treatment of symptomatic conditions?

2 MR. WHITLOCK: Objection to the form.

3 A. I think that's a reasonable statement. All I can  
4 say is, I understand and I agree.

5 Q. Isn't one of the fundamental precepts of  
6 preventive testing is that one should avoid doing more  
7 harm than benefit?

8 A. That's correct. I agree.

9 Q. Doesn't screening all comers in an asymptomatic  
10 population mean that you increase the number of false  
11 positives each of which comes with an obligation to  
12 follow-up?

13 MR. WHITLOCK: Objection to the form.

14 A. It is a theoretical question. I understand what  
15 you are saying. It is not related to my report, though.

16 Q. Why not, why do you say that it is not related to  
17 your report?

18 A. Because I did not assess the proposed  
19 biomonitoring program in any detail. I just assessed  
20 the general appropriateness of the proposal.

21 Q. What's the distinction that you are drawing? So  
22 I mean, Doctor Ducatman advanced a proposed medical  
23 monitoring program in his reports. You read those  
24 reports and you basically say at a high level, you are

1 giving those recommendations your stamp of approval; is  
2 that correct?

3 A. It is a stamp of approval in general terms. I  
4 have not evaluated those in detail, because that was not  
5 what I was asked to do. It says here in the first  
6 paragraph on Page 1, we have been through that, and so I  
7 have not essentially dissected his proposal and applied  
8 those principles. I would assume that he did. I think  
9 you should ask Doctor Ducatman about it.

10 Q. Let me take it from the other direction. While  
11 you did not dissect Doctor Ducatman's proposal, on the  
12 other side of the coin, you, yourself, have not created  
13 or proposed a particular medical monitoring program for  
14 PFOA exposed populations; is that correct?

15 A. That is correct, because I was not asked to do that.

16 Q. At any time?

17 A. I mean all I was asked to do is here in that  
18 paragraph on Page 1.

19 Q. I mean look, I don't want to, you know, beat  
20 around the bush. You have not done this with respect to  
21 any population anywhere in the world, have you?

22 A. Done what?

23 Q. You have not constructed, developed, specified,  
24 created a medical monitoring program for PFOA exposed

1 individuals?

2 A. I have not done that.

3 Q. I thought we were communicating, I just wanted  
4 to make sure?

5 A. One thing is you know the principles. One thing  
6 you know the risk assessment and the exposure related  
7 outcomes. I didn't consider that to be my task to do.  
8 That's why I am asking the way that I am.

9 MR. WHITLOCK: Counsel, can we go off the  
10 record?

11 MR. WOLFF: Sure.

12 THE VIDEOGRAPHER: The time is 3:34. We are  
13 off the record.

14 (Discussion off the record)

15 THE VIDEOGRAPHER: The time is 3:40. We are  
16 back on the record.

17 Q. Let me just see if I understand this. Is it your  
18 opinion that medical monitoring is warranted based on  
19 exposure to PFOA, but that you have not specifically  
20 undertaken to determine whether any particular tests or  
21 diseases for which Doctor Ducatman proposes to medically  
22 monitor or warranted?

23 A. I think that is correct interpretation.

24 Q. You prepared a case in chief, expert report in

1 support of the plaintiffs in PFOA litigation in New  
2 Hampshire; is that correct?

3 A. I did.

4 Q. That report is dated June 22, 2018; is that  
5 correct?

6 A. I believe that is the correct date.

7 Q. You prepared your case in chief report for the  
8 New Hampshire litigation approximately six weeks before  
9 your rebuttal report in this matter; is that correct?

10 A. That's correct. So they are quite similar.

11 Q. My question is, did you use your case in chief  
12 report in the New Hampshire litigation as a model for  
13 your rebuttal report in this matter?

14 A. I would not say I used it as a model, but I used  
15 my wordings here and there because the evidence that I  
16 am relying upon is the same and may have been updated  
17 with, some, you know, recent publications that came out  
18 after the June date.

19 Q. Do you agree the majority of your rebuttal report  
20 in this matter is the same as your case in chief report  
21 in the New Hampshire litigation?

22 A. I have not checked to which extent they are the  
23 same. I would say they are very similar because the  
24 situations are similar, but in that case there were no

1        rebuttals I needed to confront.

2            Q.    Your case in chief report in New Hampshire was  
3        not prepared to rebut another expert's opinion?

4            A.    That's correct.

5            Q.    Your case in chief report in the New Hampshire  
6        litigation was not prepared to support any opinion by  
7        Doctor Ducatman in that matter, true?

8            A.    I don't think he was involved in that case.

9            Q.    He is not.

10          A.    Okay.    So that explains that.

11          Q.    On Pages 44 and 45 of your report in this matter,  
12        you discuss diabetes; is that correct?

13          A.    That's correct.

14          Q.    Diabetes is not an endpoint that Doctor Ducatman  
15        is proposing to medically monitor for, is it?

16          A.    I don't remember so; but it is worthwhile  
17        considering.

18          Q.    You also discuss obesity in your report; is that  
19        correct?

20          A.    That's correct.

21          Q.    At the top of Page 55, you write that monitoring  
22        of body weight and BMI is essential in preventing  
23        cardiovascular disease recommendations for identifying  
24        subjects for possible obesity interventions have been

1 recently published; is that correct?

2 A. That's correct.

3 Q. Doctor Ducatman is not proposing that obesity  
4 be medically monitored as an endpoint, is he?

5 A. I don't remember, but you will see the references  
6 here are from 2018. So maybe he did not know the  
7 references at the time that he wrote this report.

8 Q. Monitoring BMI, monitoring body weight and  
9 monitoring for obesity is something that happens as a  
10 matter of routine medical care, virtually any time a  
11 patient walks into a clinical setting; is that true?

12 A. I don't know because there may be people not  
13 covered by this kind of service. I can't say that this  
14 is true in general.

15 Q. I don't mean to be flippant when I say this, but  
16 I must say from the moment I was born I was weighed and  
17 any other time I have ever stepped in a doctor's office,  
18 I am asked to takeoff my shoes by the nurse and get on  
19 the scale and be weighed. So is that your experience,  
20 too?

21 A. But I still seem to remember that Doctor Ducatman  
22 said that part of the medical monitoring was for the  
23 patient or the subject to step on the scale. So he is  
24 still saying this should be part of it and I agree.



1 Q. Stepping on the scale is no different than what  
2 most patients experience when they go in for a routine  
3 checkup, right?

4 A. If they do. So I have to side with Ducatman that  
5 he designed a program that I think is appropriate. Of  
6 course, height and weight is part of that. You cannot  
7 just exclude it because you think that people will go  
8 for a medical checkup because they may not.

9 Q. On Page 62, you also describe monitoring  
10 procedures for bladder cancer and prostate cancer; is  
11 that correct?

12 A. Right.

13 Q. Doctor Ducatman is not proposing medical  
14 monitoring for bladder cancer or for prostate cancer, is  
15 he?

16 A. I think you are correct. He is not.

17 Q. Well, why didn't you limit your opinions in your  
18 rebuttal report to the endpoints that Doctor Ducatman is  
19 proposing to medically monitor for?

20 A. It was because I was not asked to do that. I  
21 mean, if we were going to compare ideas and proposals  
22 for medical monitoring, I would have to share the  
23 references that I was familiar with and that I had  
24 picked up. Also, those after he completed his report

1 and we did not do that. It is clear that when I refer  
2 to that, let's say, Swedish study on prostate cancer, he  
3 may not have picked that up. And so we may deviate a  
4 little bit. That is only natural.

5 Q. Just to be fair and to state the obvious, the  
6 scope of your report goes beyond the scope of what  
7 Doctor Ducatman is proposing to medically monitor for;  
8 is that true?

9 A. That is not correct. My report focuses on the  
10 elevated exposure and elevated risks of what they are  
11 and that can of course be used as part of a discussion  
12 and decision on medical monitoring but that was not my  
13 task.

14 Q. What was your task?

15 A. To review the exposure associated risks of  
16 adverse health effects.

17 Q. Your rebuttal report was not targeted solely at  
18 the defense experts opinion, was it?

19 A. Not solely. Because I was looking at, I think  
20 you call it general causation. It was focused on what  
21 do we know about the adverse health effects.

22 Q. A general causation analysis was really the  
23 primary focus of your report?

24 MR. WHITLOCK: Objection to form.

1           A. We would have to ask a counsel about that. I was  
2 given the task as described here and clearly I spent  
3 more space on evaluating the strength of the evidence  
4 than I did on rebutting some report. I think I spent a  
5 page on each of them. That is really a very small  
6 component.

7                   MR. WOLFF: Let's go off the record for a  
8 moment.

9                   THE VIDEOGRAPHER: The time is 3:49. Going  
10 off the record.

11                   (Discussion off the record)

12                   THE VIDEOGRAPHER: Back on the record. The  
13 time is 3:50.

14                   MR. WOLFF: Thank you. I have no further  
15 questions at this time.

16                   CROSS EXAMINATION

17           BY MR. WHITLOCK:

18           Q. Doctor Grandjean, I just have one question for  
19 clarification purposes. Towards the end there of  
20 Mr. Wolff's questioning, there was several questions  
21 about the scope of your testimony in regards to the  
22 design of Doctor Ducatman's medical monitoring program.  
23 There was a question asked about your, the level of your  
24 review of the tests and the record will speak for

1     itself, but I believe disease endpoints as recommended  
2     by Doctor Ducatman in his medical monitoring program. I  
3     just want to be clear, on Pages 27 through 62, I  
4     believe, of your report, Section 8, which is titled  
5     Adverse Health Effects at Individual Endpoints, did you  
6     in that section of your report evaluate the  
7     epidemiological literature along with the toxicological  
8     evidence as you discussed throughout your deposition  
9     today in regards to the specific disease endpoints that  
10    Doctor Ducatman chose for his medical monitoring  
11    program?

12                 MR. WOLFF: Objection to form.

13                 A. I believe I covered the endpoints that he  
14     assessed and considered appropriate for his monitoring.  
15     I think I covered all of them.

16                 MR. WHITLOCK: Thank you. That is my only  
17     question.

18                 MR. WOLFF: We are done. Thank you.

19                 THE VIDEOGRAPHER: The time is 3:52. We are  
20     going off the record. This is the end of today's  
21     deposition of Philippe Grandjean.

C E R T I F I C A T E

I, Philippe Grandjean, having read the foregoing transcript of my testimony, do hereby certify under the pains and penalties of perjury. The same contains a true and accurate record of my answers to the questions herein set forth, together with correction pages, if any, attached.

---

PHILIPPE GRANDJEAN

Subscribed and sworn to before me  
this \_\_\_\_ day of \_\_\_\_\_, 2018.

---

NOTARY PUBLIC

C E R T I F I C A T E

Commonwealth of Massachusetts  
Suffolk, ss.

I, William M. Jackson, a notary public in and for  
the Commonwealth of Massachusetts, do hereby certify:

That Philippe Grandjean, the witness whose  
testimony is hereinbefore set forth, was duly sworn by  
me, and that such testimony is a true and correct  
transcription of my stenotype notes taken in the  
foregoing matter, to the best of my knowledge, skill and  
ability.

I FURTHER CERTIFY that the foregoing transcript  
is a true and correct record of the testimony given by  
the said witness at the time and place specified  
hereinbefore.

I FURTHER CERTIFY that I am neither a relative  
nor employee of nor counsel for any of the parties, nor  
am I financially interested directly or indirectly in  
the outcome this action.

IN WITNESS WHEREOF, I have hereunto set my hand  
and Notarial Seal this 13th day of November, 2018.



William M. Jackson  
Notary Public

My Notary Commission expires: October 26, 2018.

\*\*\*\*

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**ERRATA SHEET**

2

**VERITEXT LEGAL SOLUTIONS**

3

**330 OLD COUNTRY ROAD**

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**MINEOLA, NY 11501**

5

**800.727.6396****CASE: SULLIVAN VS. SAINT-GOBAIN PERFORMANCE****DEPOSITION DATE: OCTOBER 10, 2018**

6

**DEPONENT: PHILIPPE GRANDJEAN**

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**PHILIPPE GRANDJEAN**

21

**SUBSCRIBED AND SWORN TO BEFORE ME**

22

**THIS \_\_\_\_\_ DAY OF \_\_\_\_\_, 20\_\_\_\_.**

23

24

**(NOTARY PUBLIC)****MY COMMISSION EXPIRES:**

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[3a - adults]

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[answer - association]

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF SEPTEMBER 1, 2016. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

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COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

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